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ON POISONS

IN RELATION TO

MEDICAL JURISPRUDENCE AND MEDICINE.

BY

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TRUDITUR DIES DIE.

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PREFACE TO THE THIRD EDITION.

But a few words are required to introduce this volume. It is based on the two previous editions; but the complete revision, rendered necessary by time, has converted it into a new work. Like the preceding editions, it is not intended to be a complete history of Poisons and Poisoning. Its size will at once show that it can only comprise a small portion of a vast subject. It is, in fact, offered simply as a Manual for the use of students and practitioners in Law and Medicine, and therefore it has been kept within very moderate limits. Some substances described as poisons do not find a place here, simply for want of space, and on this, as on former occasions, I have exercised a freedom of selection with a special view to practice. As a rule I have omitted a description of those poisonous substances which have not hitherto given rise to investigations before our legal tribunals, and which are otherwise of little interest to the profession. In short, as the reader will perceive, the subject of poisons has been treated only in relation to Medical Jurisprudence and Medicine, and as fully as the space placed at my disposal would permit.

The whole work has been remodelled, and a number of illustrations have been introduced. Some chapters have been omitted, some divided, and others introduced, in accordance with the changing aspect of toxicological science. *Quod hodiè exemplis tuemur mox inter exempla erit.*

A. S. T.

15 St. James's Terrace,
Regent's Park, May, 1875.

54076

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83. Leaves of œnanthe crocata, 710
84. Leaves of æthusa cynapium, 713
85. Seeds of æthusa cynapium, 713
86. Small leaf of aconitum napellus, 716
87. Seeds of aconitum napellus, 716
88. Root of aconite, 717
89. Root of horseradish, 717
90. Leaf of belladonna, 727
91. Seeds of belladonna, 731
92. Crystals of pure atropia, 733
93. Imperfect crystals of sulphate of atropia, 733
94. Seeds of lobelia, 736
95. Seeds of datura stramonium, 741
96. Seeds of datura alba, 741
97. Leaf of datura stramonium, 742
98. Crystals of datura, 743
99. Leaf of laburnum, 746
100. Seeds of laburnum, 746
101. Yew leaves and fragments, 749
102. The calabar or ordeal bean, 756
103. Leaves of foxglove, 762
104. Seeds of foxglove, 762

ON POISONS.

CHAPTER I.

Toxicology—Meaning of the term poison—Medicines and poisons—Large and small doses—Medical definition—Legal definition—Administration of poison—Noxious and destructive things.

By Toxicology (derived from τοξικὸν, *poison*, and λόγος, *discourse*) we are to understand that branch of medical science which relates to the history and properties of poisons, and of their effects upon the living body. This subject is commonly regarded and treated as a part of Medical Jurisprudence; but the number and importance of the facts connected with poisons which have been accumulated of late years, have justly contributed to raise Toxicology to the rank of a distinct science. To the physician, the pathologist, and the medical jurist, a knowledge of the subject is of great importance: for cases are continually presenting themselves in which a practical application of the principles of this science is demanded; as, for example, in the treatment of a person laboring under the effects of poison, in drawing a clear distinction between changes produced in the body by disease and those caused by poison, or finally, in aiding the criminal law in convicting those who have been guilty of the crime of poisoning.

Definition.—A Poison is commonly defined to be a substance which, when administered in *small quantity*, is capable of acting deleteriously on the body; in popular language this term is applied only to substances which destroy life in small doses. This view of the nature of a poison is too restricted for the purposes of medical jurisprudence. It would obviously exclude numerous compounds, the poisonous properties of which cannot be disputed; as, for example, the salts of copper, tin, zinc, lead, and antimony, which, generally speaking, act only as poisons when administered in large doses. It must not be supposed, however, from this statement, that the compounds of these metals are innoxious in small doses. It is usually said that a poison in a small dose is a medicine, while a medicine in a large dose is a poison. Thus strychnia may be regarded either as a medicine or a poison. In a dose of half a grain it has destroyed the life of an adult, and is a poison, while the sixteenth part of a grain has been taken by an adult safely and beneficially as a medicine. But regard must also be had to the age of the patient; thus, the last-mentioned quantity, which was safely taken by an adult, has acted as a poison, and destroyed the life of a child four years of age. A person may die either from a large dose given at once, or from a number of small doses given at such intervals that the system cannot recover from the effects of one before another is administered. In cases of lead-poisoning it is a well-known fact that a quantity of carbonate of lead so small as to be scarcely appreciable to tests, may, by its daily introduction into the system through water or other articles of food, produce symptoms of chronic poisoning, which, although different in their nature and progress, are not less fatal than those which are produced by a large dose of a salt of lead.

In legal medicine, it is difficult to give such a definition of a poison as shall be entirely free from objection. Perhaps the most comprehensive which can be suggested is this: "A poison is a substance which, when absorbed into the blood, is capable of seriously affecting health or of destroying life." There are various channels by which poisons may enter the blood. Some are in the form of gases or vapors: these operate rapidly through the lungs; others are liquid or solid, and these may reach the blood either through the skin or through a wound, but more commonly through the lining membrane of the stomach or bowels, as when they are taken or administered in the ordinary manner. The latter chiefly give rise to medico-legal investigations. Some substances act as poisons by any one of these channels: thus arsenic is a poison, whether it enters the blood through the lungs, the skin, or the stomach and bowels; but such poisons as those of the cobra, the viper, of rabies, and of glanders, appear to affect the body chiefly through a wound. When introduced into the stomach, some of these animal poisons have been found to be inert.

In adopting the above definition of a poison in a medical sense, it is proper to remark that there are some substances which are regarded as poisons, although absorption into the blood does not appear to be absolutely necessary to their action. The mineral acids and alkalies belong to this class of bodies. They are corrosive poisons; they operate injuriously by causing the destruction of living parts; and whether applied to the skin, the stomach, or (in the form of vapor) to the air-cells of the lungs, they destroy life chiefly by the local changes to which they give rise, and the inflammation which is a consequence of their action.

In reference to the *medical* definition of a poison, it is necessary to observe that the law does not regard the manner in which the substance administered acts. If it be capable of destroying life or of injuring health, it is of little importance, so far as the responsibility of a prisoner is concerned, whether its action on the body is of a mechanical or chemical nature, and whether it operates fatally by absorption into the blood or not. Thus a substance which simply acts mechanically on the stomach or bowels, such as glass or iron filings, may, if wilfully administered with intent to injure, involve a person in a criminal charge, as much as if he had administered arsenic or any of the ordinary poisons.

We may now consider what the law strictly means by the act of poisoning. If the substance criminally administered destroys life, whatever may be its nature or mode of operation, the accused is tried on a charge of murder or manslaughter, and the duty of a medical witness consists in showing that the substance taken was the certain cause of death. If, however, death is not the consequence, then the accused may be tried for the attempt to murder by poison. (24 and 25 Vic., c. 100, s. 11, Aug. 1861.) The words of this statute are general; they embrace all kinds of substances, whether they are popularly or professionally regarded as poisonous or not, and they leave the question "What is a poison?" to depend upon the medical evidence adduced. In order to include all substances of an injurious nature, although they may not be strictly speaking poisons, the words "*destructive* or *noxious thing*" are employed. Hence on these occasions, a medical witness must be prepared to prove that the substance, if not a poison in the ordinary meaning of the term, was really a destructive or noxious substance, *i. e.*, injurious to health.

In March, 1874, a man was charged, under the Adulteration Act, with selling lozenges containing powdered glass and blue starch. Dr. Bernays, who gave evidence respecting the adulteration, was asked whether the glass was injurious to health. His reply was that it was not, unless taken in large quantity by children, and here the quantity was very small. The case was dismissed. Prosecutions under the Adulteration Act (35 and 36 Vic., cap. 74, Aug. 1872) have given rise to many inquiries respecting the poisonous or injurious nature of a variety of substances mixed with articles of food.

The term adulteration, as it is used in this act, includes, also, any mixture of substances not injurious to health, which increases the weight or bulk of things sold, as of water with milk, or chiccory with coffee. Dr. Letheby found 40 per cent. of iron filings and 19 per cent. of silica in the form of fine sand in certain kinds of tea. Prussian blue, French chalk, and yellow coloring matter have been detected in green teas; red oxide of iron in anchovy sauce and paste; and red lead in snuff. In all these cases the analyst must be prepared to state whether such substances are or are not poisonous and injurious to health.

In reference to the crime of poisoning, the words of the statute are as follows: "Whosoever shall administer, or cause to be administered to or taken by any person, any poison, or *other destructive thing*, with intent to commit murder, shall be guilty of felony."

Whether the administering be followed by any bodily injury or not, the act is still a felony, provided the *intent* has been to commit murder. The attempt to administer, or the attempt to cause to be administered to, or to be taken by any person, any poison or *other destructive thing*, with the like intent, although no bodily injury be effected, is also a felony (s. 14). If any doubts formerly existed whether the *external* application of poisons, *e. g.*, by wounds or ulcerated surfaces, would be included in the words "administering or taking," they are now entirely removed by the Criminal Law Consolidation Act (Aug. 1861). The 22d section specially applies to such an offence, and the 15th section provides that "Whosoever shall, by any means other than those specified in any of the preceding sections of this act, attempt to commit murder, shall be guilty of felony." Mr. Greaves justly remarks, with regard to this important addition to the statute law, that "the malicious may now rest satisfied that every attempt to murder which their perverted ingenuity may devise, or their fiendish *malignity suggest*, will fall within some clause of this act, and may be visited with penal servitude *for life*." (*Notes on Crim. Law Consolidation*, p. 49.) Under section 22 of this statute, in reference to attempted poisoning, some offences are comprised, which formerly escaped punishment: "Whosoever shall unlawfully apply or administer to, or cause to be taken by, or attempt to apply or administer to, or attempt to cause to be administered to or taken by any person, any chloroform, laudanum, or other stupefying or overpowering drug, matter, or thing, with intent, in any of such cases, thereby to enable himself or any other person to commit, or with intent, etc., to assist any other person in committing any indictable offence, shall be guilty of felony."

Poison is not always administered with intent to murder. On many occasions it has been mixed with food, and thus administered with a view to injure or annoy a person. Cantharides have been thus frequently given, and in one instance (Nov. 1859) eight members of a family suffered from severe symptoms of poisoning by reason of the wanton administration of this drug. In April, 1860, several members of a family suffered from severe sickness, as a result of tobacco having been put into water contained in a tea-kettle; and tartar emetic has been in some cases dissolved in beer or other liquids as a mere frolic, without any proved or probable intention on the part of the offender to destroy life. Hitherto, when the intent to murder has not been proved, the offender has escaped, although great bodily injury may have been done by his wanton or malicious act. Sections 23, 24, and 25 of the Consolidation Act, c. 100, provide for this omission.

"23. Whosoever shall unlawfully and maliciously administer to, or cause to be administered to or taken by any other person, any poison or *other destructive* or *noxious thing*, so as thereby to endanger the life of such person, or so as thereby to inflict upon such person any grievous bodily harm, shall be guilty of felony.

"24. Whosoever shall unlawfully and maliciously administer to, or cause to be administered to or taken by any other person, any poison or other destructive or noxious thing, with intent to injure, aggrieve, or annoy such person, shall be guilty of a misdemeanor.

"25. If, upon the trial of any person charged with the felony abovementioned, the jury shall not be satisfied that such person is guilty thereof, but shall be satisfied that he is guilty of the misdemeanor abovementioned, then and in every such case the jury may acquit the accused of such felony, and find him guilty of such misdemeanor."

CHAPTER II.

ABSORPTION OF POISONS—CHANNELS OF ENTRANCE AND EXIT—ENTRANCE INTO AND DIFFUSION BY THE BLOOD—ACTION BY INJECTION INTO THE BLOOD OR WOUNDS—ABSORPTION BY THE UNBROKEN SKIN—DEATH FROM CONTACT WITH DISEASED SKIN—HYPODERMIC INJECTIONS—POISONING BY HYPODERMIC INJECTIONS.

POISONS may enter the body by various channels. The aerial poisons, including gases and vapors, enter by the air-passages during the act of breathing. Metallic and metalloidal poisons, which are capable of assuming the gaseous form or of being diffused in a fine dust, may also find their way into the body by the lungs. Arsenic, antimony, and phosphorus, in their combinations with hydrogen, may act as aerial or gaseous poisons. In the section on arsenical poisoning, will be found described some cases in which arsenuretted hydrogen has thus proved fatal.

There are three conditions connected with the action of poisons on the body which require special notice: 1, absorption and diffusion by the blood; 2, elimination by the fluid secretions and excretions; and 3, their temporary deposition in the soft organs and tissues. These processes go on simultaneously. As soon as a poison has been carried into the blood by absorption, one portion of it is thrown off by the fluids of the body, and another portion is deposited in the tissues. In reference to liquid poisons, they are simply diffused through the soft organs by the blood, and those which are in the state of gas or vapor, are chiefly eliminated from the lungs.

The cases which are supposed to be adverse to the theory of absorption are so few that they may be disregarded. The apparent difficulties connected with them will probably disappear by further scientific researches. Thus it has been suggested that the effects produced by serpent poison are too rapid to be accounted for by absorption; but in the numerous experiments performed on this subject by Drs. Fayrer and Brunton, there was always a sufficient lapse of time for the poison to find its way into the blood before any symptoms were manifested. They state that there are few, if any, instances on record of death from the fresh poison of the cobra in less than *half a minute*, the time in which the dried poison killed a guinea-pig. (*Proc. R. S.*, Jan. 1874, p. 78.)

The channels of entrance may be thus enumerated: 1, the blood-vessels, including wounds; 2, the skin and cellular membrane; 3, the air-passages and lungs; 4, the stomach; and 5, the intestines. The channels of exit by which poisons are eliminated or excreted from the body, may be named in the following order: 1, the urine; 2, the bile; 3, the milk; 4, the saliva; 5, mucous secretions; 6, serous secretions;

7, the perspiratory fluid. The organs or tissues in which they undergo an intermediate deposit, but from which they are ultimately expelled, may be thus enumerated: 1, the liver; 2, the kidneys; 3, the spleen; 4, the heart; 5, the lungs; 6, the muscles; 7, the brain; 8, the fat; 9, the bones.

Cases of poisoning in a medico-legal point of view, are commonly confined to those in which the poison has passed by the mouth into the stomach. The mucous membrane of all parts of the body is absorbent; hence these agents may operate equally as poisons by contact with the mucous membrane of the nose, eye, vagina, or rectum.

Absorption and Diffusion.—As a general rule, whatever may be the surface or texture to which a poison is applied, it is sooner or later absorbed and circulated with the blood before it begins to manifest its effects. Liquid poisons when swallowed (if we except substances which have a local and corrosive action) are more rapidly absorbed than those which are solid. Soluble poisons are absorbed more rapidly than those which are insoluble. Some solid substances which are but little soluble (arsenious acid) are, however, very soon absorbed in sufficient quantity to produce well-marked symptoms. Others, which are not very soluble in water, may become dissolved in the acid mucous secretions of the stomach, and they are then readily carried into the blood. The carbonate of lead, white precipitate, and arsenite of copper (Scheele's green), which are insoluble in water, are thus rendered sufficiently soluble for absorption through the mucous membrane of the stomach.

Poisons are absorbed by the bloodvessels of the part to which they are applied. The coats of these vessels are thin and porous, and readily imbibe any liquid placed in contact with them, or any solid which is soluble in the surrounding fluids, as the serous fluid of the cellular tissue, or in the mucous fluid of the mouth, stomach, and intestines. Osmosis or a penetration of the coats then takes place with great rapidity, and the liquid or gaseous poison, when once in the blood, is rapidly carried by it to all parts of the body.

If the minute vessels are cut through as in a wound, the poison at once mixes with the blood, and is circulated with it. This is not absorption, but injection, and the effects of all poisons are thus displayed with the greatest rapidity and intensity.

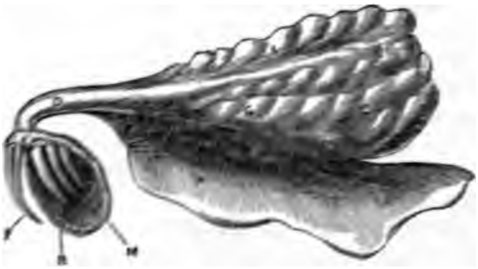
Entrance into the blood as the result of absorption or injection, is a condition necessary to the action of a poison. This was the doctrine long since taught by Magendie, and, in more recent times, it has received confirmation from the experiments of Dr. Blake, M. Bernard, Müller, and other physiologists. M. Bernard has demonstrated that until a poison has reached the arterial capillary system, as the result of absorption, however deadly it may be, there is no symptom indicative of poisoning. Strychnia or prussic acid applied directly to the brain, spinal marrow or nerves, produces no effect, or only a slight local action after some time, but when a portion of either of these poisons is carried by absorption into the arterial capillary system, the symptoms of poisoning appear. (*Leçons sur les Effets des Substances Toxiques*, p. 47. Paris, 1847.)

Hence it follows that whatever prevents the entrance of a poison into the blood, arrests the symptoms of poisoning. It had been already proved by the experiments of Sir R. Christison, that extract of nux vomica, put into a wound in the paw of a dog, produced no symptoms of poisoning when a ligature was placed tightly around the leg; but when this was relaxed, tetanic symptoms were soon produced, and thus poisoning might be made artificially intermittent. These results have been confirmed by the more recent investigations of Dr. Fayrer on the poison of the cobra de capello. He found that fowls bitten by this serpent in the leg or wing were saved by immediate amputation, *i. e.*, by the removal of the bitten part, within a few seconds. A dog bitten in a fold of skin raised for this purpose, and immediately excised by a clean sweep of the scalpel, also escaped. The rapidity of absorption was, however, on some occasions so great, that symptoms were observed within a few seconds after the bite. Another fact was brought out by these experiments, which has an immediate bearing upon the treatment of cases of poisoning by snake-bite, namely, the extreme difficulty of completely arresting the circulation in a limb by a ligature. A fowl having had a ligature tightened around its thigh with the greatest amount of tension a man's hand could exert, was bitten below it by a cobra. The limb of the fowl was probably more thoroughly strangulated than a human limb could possibly be by any kind of tourniquet, and yet in twenty-three minutes the fowl began to show symptoms of poisoning, and in twenty-one minutes more it died. This fact proves that a ligature cannot altogether keep out the poison, but it may retard its operation and give time for treatment, and the result shows that the most potent and insidious of all poisons operates as such on the body, only after it has been absorbed and diffused by the circulation. Although the cobra poison is rapidly absorbed, the effects are not immediate. Dr. Macbeth found that, in fowls, the interval between the bite and the symptoms was from nine and a half to fifteen minutes; in dogs, from an hour and eight minutes to two hours and ten minutes (*Australian Medical Jour.*, Nov. 1871.) In an experiment with cobra-poison made by Dr. Pavy and myself, the symptoms in a rabbit came on a quarter of an hour after its introduction into the cellular membrane.

1. *The Bloodvessels, including Wounds.*—This mode of entrance into the body has rather a physiological than a medico-legal interest. When a poison is introduced directly into the *blood*, either by injection into a vessel or by a wound, it will be understood that its effects are rapidly produced. Sir R. Christison found that when the muriate of conia was injected into the femoral vein of a dog, he was unable, with his watch in his hand, to notice any appreciable interval between the moment at which it was injected and that in which the animal died. The interval did not exceed three, or at most four, seconds. Prussic acid and strychnia act almost instantaneously under these circumstances. Dr. Fayrer found that when the cobra poison was injected into the jugular vein of an animal, the action of the heart was at once arrested. The heart was not paralyzed, but thrown into a state of tetanic contraction from excessive stimulus.

I am indebted to Dr. Fayrer for the annexed diagram of the poison-gland of a poisonous snake. The gland is incased in a capsule, and is partially covered by fibres of muscle (the masseter) whose action in closing the jaw at the same time compresses the gland and squeezes the poison through the duct into the perforated or grooved fang, whence it issues at F (see Fig. 1).

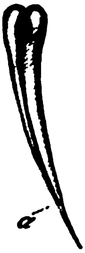
FIG. 1.



Poison glands, duct and fang in situ. L, lobe of gland; D, duct; F, fang; GG, gland; M, mucous capsule of fang; R, reserved fangs; AA, fascia or membrane covering the glands.

There is probably no instrument so perfectly constructed as the tooth of a venomous serpent for the introduction of liquid poison by means of a wound. It is such as to insure its rapid absorption and diffusion through the body of the bitten animal. The tooth is curved, and is grooved or channelled on the front or convex side, as if it were folded upon itself. It is through this channel the poison is injected into the deep and curved puncture made through the skin (Fig. 2). The point of the tooth is solid and finely sharpened. Mr. C. Tomes informs me that he has found this portion of the tooth to consist of pure enamel of the hardest kind. The channel or groove, through which the poison is discharged, terminates on the front of the tooth (*a*, Fig. 2) at a short distance above the point. By this arrangement its sharpness is always preserved. The poison is injected into the wound, and therefore into the blood, in the act of the serpent inflicting the bite. As a result of the curved form of the tooth the wound is valvular, so that it retains the poison, and the blood which escapes is small in quantity.

FIG. 2.



Poison-fang of the serpent, grooved in front. *a*, aperture for the discharge of poison.

The curara and other arrow poisons of savage nations operate through wounds. Arsenic and mineral poisons may also operate in a similar manner. In order to test this question Mr. Swan introduced a portion of arsenic into a wound in the back of a dog. Vomiting came on in two hours, and the animal died in six hours. The mucous membrane of the stomach and intestines was found inflamed. (*Action of Mercury*, p. 33.)

2. *The Skin and Cellular Membrane.*—The process of absorption is modified, not only by the state of the poison, but by the nature of the surface or the texture of the part to which it is applied. Every substance acting as a poison must pass into the body, either through the

skin or through the mucous membrane, and there are many circumstances which may favor or retard absorption by either of these channels. Thus in the skin covered with cuticle the process of absorption is slow. The membrane which is here interposed between the poison and the coats of the bloodvessels is not readily penetrated. When, however, the cuticle is removed, and the minute vessels are exposed either on the surface of the cutis or as a result of granulation, then the penetration takes place readily.

The unbroken skin is naturally covered with an oily or sebaceous secretion, and this is found to favor the entrance of all solid substances that are in fine powder, or that are left in a finely divided state on the cuticle, by the evaporation of their aqueous solutions; but on the other hand it retards or prevents the introduction of those poisons which are dissolved in water. M. Roussin has experimented on himself with baths of iodide of potassium, and he found that not a trace of the substance was absorbed by the sound skin. (*Ann. d'Hygiène*, 1867, vol. 2, p. 194.) All fatty or oily matters, or liquids which will dissolve them, such as chloroform, ether, or alcohol, favor absorption through the cuticle. It has been long known that mercury mixed with lard as mercurial ointment, when rubbed into the skin is readily absorbed, and produces salivation and other effects of mercurial poisoning. A combination of oleic acid with oxide of mercury has been found to operate more powerfully and rapidly than the ordinary compound with lard. Dr. Neumann inferred, from his experiments on metallic mercury, that the mercurial globules passed by inunction into the hair-sheath, then into the bulb; into the superficially opening sebaceous glands, and into the upper part of the sweat-glands. The metal is, no doubt, dissolved before entering the bloodvessels.

Opium and other powerful drugs, combined with alcohol or saponaceous liquids, also readily penetrate the cuticle by friction. I have known symptoms of incipient narcotism to be produced by the application of an ordinary soap liniment containing tincture of opium to the unbroken skin. Aconite, belladonna, and other powerful poisons, used in the form of ointment or tincture, are also absorbed, and produce their usual effects. The oily or sebaceous matter which is diffused over the unbroken skin is sufficient to cause the penetration of a solid poison, provided it is in the form of a fine powder, or left as such by the evaporation of a solvent. Roussin has demonstrated this on his own person (*Op. cit.*, p. 196), and it would be well if medical men who use strong alcoholic solutions of corrosive sublimate or other poisons for the treatment of diseases of the skin, would bear this fact in mind.

According to Sir James Paget, an abrasion or wound of the skin is not necessary for absorption in reference to the morbid fluids occasionally met with in a dead body. He cites, in proof of this statement, his own case, in which his life was placed in great jeopardy. (*Lancet*, 1871, vol. 1, pp. 735, 805, and vol. 2, p. 537.) Probably long contact with the unbroken skin may here compensate for the comparative impermeability of the cuticle.

I have elsewhere reported the case of a furrier who died from chronic poisoning by mercury, as a result of handling and packing skins impregnated with the dry nitrate of this metal. (*Guy's Hosp. Rep.*, 1864, p. 173.) The frequent handling of lead or pewter has given rise to lead colic. The fine particles of metal which are rubbed off and adhere to the skin, are converted by the sebaceous secretion and perspiration into soluble salts of lead which are rapidly absorbed. The late Dr. Todd noticed that men who were much occupied in cleaning pewter pots by rubbing them with their naked hands, were subject to painter's colic.

The use of hair-washes containing salts of lead dissolved, has given rise to paralysis, and other symptoms of lead-poisoning. Absorption has taken place as a result of frequent use, although the skin was unbroken.

Some mineral poisons, in the state of fine powder, such as white arsenic, white lead, and emerald green, have produced the usual effects of poisoning by these substances, chiefly in cases where there was a want of cleanliness—the powder being allowed to collect and remain in the folds of the skin, and there to cause irritation and inflammation, followed by the absorption of the mineral. (*Ann. d'Hyg.*, 1859, vol. 2, p. 49.) All the symptoms of arsenical poisoning, although not appearing for two or three days, have been witnessed in the human body in those cases in which powdered arsenic has been used as a depilatory. (See *Ann. d'Hyg.*, 1846, vol. 2, p. 157.)

The effects of arsenic on the unbroken skin was a subject of inquiry at the trial of Madeline Smith (Edinburgh, 1857). The reader will find in another chapter some remarks in reference to this case (p. 69). Accidents frequently arise from the use of arsenic by shepherds, for the purpose of destroying the fly in sheep. Two shepherds were engaged in sheep dipping for nine hours; the liquid used being a mixture of white arsenic in a solution of carbonate of potash. On the following day both were attacked with similar symptoms. One of the men, when seen on the fourth day from the dipping, had the skin of the scrotum covered with eczema rubrum, resembling the appearance seen after vesication and separation of the cuticle in patches. There were also vesicles on the thighs. There was slight febrile disturbance, with intense thirst, which no amount of liquid could allay. In a few days the man recovered. (*Lancet*, Sept. 12, 1857, p. 282.) Dr. Watson, who relates this case, states that the man had previously had similar eruptions from the use of the dipping composition. Other shepherds, who had used arsenic, had also suffered from eruptions, principally on the hands, forearms, scrotum, and thighs; and this had happened when the arsenic was used alone.

It appears that in India yellow arsenic is much used in the manufacture of shellac, and it is not unusual for eruptions of an eczematous nature to appear on the skins of those who are engaged in the manufacture. (Dr. N. Chever's *Med. Jour. for India*, p. 584.) Some facts regarding this form of poisoning will be found in the *Ann. d'Hyg.*, 1846, vol. ii, p. 131.

The destruction of life by the local application of arsenic to ulcerated surfaces has never, so far as I am aware, been resorted to by criminals.

It is obvious that, when death is not a consequence, serious injury to health may ensue, and life may be endangered.

There have been several cases within the last ten years in which persons have died from the application of arsenic and other poisons to the *diseased skin*, and unfortunately there is a great deal of popular ignorance on the subject. Whenever the skin is ulcerated or diseased, the application of a poisonous substance is followed by rapid absorption and death. A girl has lost her life by the use of a strong solution of corrosive sublimate for the cure of ringworm, and a woman has died with the usual symptoms of narcotic poisoning, in an aggravated form, from the application of morphia to an ulcerated breast.

In the *Pharmaceutical Journal* (for 1873, p. 752), a case is reported in which a man died from using a liniment consisting of thirty grains of carbolic acid dissolved in alcohol and water. He rubbed it in for the purpose of curing the itch, and it is stated that he died in an hour. In the same journal (for May 16, 1874, p. 926), another fatal case is recorded, apparently from the absorption of this poison through the cutis exposed by a burn. The deceased had been severely burned in a colliery explosion, but was going on apparently well. An ointment containing carbolic acid was applied to the burned parts on cloths, but the pain caused by it was so severe that they were removed. The man became much worse after the application, suffered from delirium, and died.

Children have died from the use of arsenic and white precipitate applied to the head, in the form of ointment, for the removal of vermin or for the treatment of ringworm. (See *Guy's Hospital Reports*, 1864, p. 220.)

In the *British Medical Journal* (May 16, 1873, p. 649), Dr. Althaus reports a case of lead-paralysis occurring in a patient who had used the subacetate of lead, in an ointment, for a sore on the thigh, daily for a month. He had used the ointment in large quantity, and kept it on the part for a long time.

Quack doctors are in the habit of using arsenical ointments freely for the removal of tumors which they call cancerous; and on two occasions I have been required to give evidence on trials for manslaughter in such cases. These irregular practitioners act upon the principle that arsenic is not a poison when applied externally, and, even when the patient dies with the symptoms of poisoning, they profess to believe that death was owing to some natural cause. MM. Chevallier and Bayard have given the details of two cases which proved fatal, owing to the application of arsenical compounds to the breasts of women. (*Ann. d'Hyg.*, 1846, vol. 2, p. 131.)

A case is reported, in *Rust's Magazine*, in which a man covered his head with arsenic in powder to act as a depilatory. He was affected with the usual symptoms of arsenical poisoning, excepting diarrhœa, and he died on the *twentieth* day. The interior of the stomach, as well as the lower part of the œsophagus, was generally inflamed. The following case, communicated to me by the late Mr. Tubbs, of Upwell, proves that arsenic, when rubbed on the skin, has decidedly a local *irritant* action. A man who was subject to piles, was in the habit of anointing himself with lard. By mistake, on one occasion, he used some white ointment containing arsenic. The next day he complained of an intolerable itching of the anus and scrotum; and, on examination, the parts were covered with pustules surrounded with an inflamed base. On examining the matter from the pustules, it was found to contain arsenious acid. Frictions of lime-water and oil were used, and the patient soon recovered.

Instances of arsenic thus destroying life, when applied externally, are by no means unfrequent. Two cases of its operating fatally in children, when applied to the skin of the head for tinea capitis, will be found in the *Annales d'Hygiène* (1830, vol. 2, 437). In both, the mucous membrane of the stomach was found inflamed, and in one extensively. Dr. Stillé quotes the following case: A woman rubbed half an ounce of arsenic mixed with gin into the heads of her children, who were affected with porrigo (scalled head). This application was followed by redness and swelling of the face. One child, two years of age, died from the effects; having suffered from purging, with paralysis of the lower limbs before death. There was no local inflammation produced. (*Med. Jur.*, 1855, p. 420; *Am. Jour. Med. Sci.*, July, 1851, p. 259.)

When the substance acts chemically on the skin, so as to soften or dissolve the cuticle and expose the cutis or cellular membrane beneath, absorption takes place more rapidly, and the usual effects of poisoning follow. Sheep-dipping liquids composed of arsenic, or arsenite of potash, with soft soap, act on the skin and dissolve the cuticle. Many years since, arsenic was used in the manufacture of stearin candles. Some deaths took place by absorption as the result of handling the materials, and this dangerous manufacture was stopped. Solutions of bichromate of potash, cyanide of potassium, and of the sulphates of copper and iron, by contact with the skin, act more or less as corrosives; they penetrate it and are absorbed, causing symptoms of poisoning among the workmen who dip their hands in these liquids. Substances like oxalic acid, not reputed to be corrosive, may also exert a strong local action on the skin, when brought frequently in contact with it. In the Museum of Guy's Hospital are two wax models of the hands of a patient who had been in the habit of cleaning pewter pots with oxalic acid. When admitted, he was very ill, having considerable febrile disturbance and complaining of severe pain in his hands. They were swollen, of a dark purple color, and apparently in a stage of inflammation approaching to gangrene. The tips of the fingers were filled with bladders of pus. The models represent accurately the appearances described. There was no other cause for this condition of the hands than that of the constant wetting of them with a strong solution of oxalic acid.

When the cuticle only is removed, as by a blister, and the surface of the cutis is laid bare, absorption takes place rapidly. In a case of scald an opiate liniment was applied by an ignorant nurse to the bare cutis. Symptoms of narcotism soon appeared, and the patient nearly lost his life.

Cases of poisoning through the skin have been called cases of "poisoning by absorption," but all cases of poisoning are the result of absorption. There is only a difference in time. As a rule, the symptoms are more slow in appearing when the poison is applied to the skin; but they resemble those produced by the poison when it enters the body by the stomach.

Poisons are rapidly absorbed when injected into the *cellular membrane* beneath the skin. This has been, of late years, adopted as a well-known mode of treatment, under the name of hypodermic injection. Orfila was the first to resort to this method in order to test the effects of poisons and their rate of absorption. He found that absorption took place rapidly, and that small quantities produced powerful effects. Thus he ascertained, by this method, that from a grain to a grain and a half of arsenic in fine powder was sufficient to kill a dog. (*Toxicologie*, 1852, 5ème ed., p. 429.) The hypodermic treatment has led to some fatal accidents, owing to this point not having been sufficiently attended to. A patient in Guy's Hospital nearly died from the injection of a small dose of atropia, which, from other trials, it had been considered safe to use. In one year, in this metropolis, there were three deaths of adults from the injection, in each case, of a grain of hydrochlorate of morphia.

Dr. Fraser found that rabbits were destroyed with very small doses of strychnia, when the poison was administered in solution by hypodermic injection. Thus from one-twentieth to one-fiftieth of a grain caused the most violent tetanic convulsions, and in a few minutes these were followed by death.

Hypodermic injection has been, in some cases, resorted to for the treatment of cases of poisoning. The introduction of poisons into wounds in the cellular membrane of animals is sometimes employed as a physiological method of testing the effects of these agents when a chemical analysis leaves their nature doubtful, or of corroborating the results already obtained by the application of chemical tests.

CHAPTER III.

ENTRANCE OF POISONS INTO THE BODY—ABSORPTION BY MUCOUS MEMBRANE—ACTION THROUGH THE AIR-PASSAGES AND LUNGS—THROUGH THE MUCOUS MEMBRANE OF THE STOMACH AND INTESTINES—ACTION THROUGH THE CONJUNCTIVA—RATE OF ABSORPTION—FATAL DOSE.

MULLER determined, by experiment, that the rapidity of absorption through membranes is in an inverse proportion to the thickness of their epithelia or surface-coverings. For this reason poisons penetrate more rapidly by absorption through the mucous membrane of the eye, mouth, and alimentary canal than through the skin, and owing to its extreme tenuity, more rapidly through the membrane of the air-cells of the lungs than through any other tissue. The effect here, when the poison is in a gaseous state, resembles that which results from the direct introduction of a poison into the blood by injection.

3. *The Lungs and Air-passages.*—For the reason above stated, the mucous membrane of the lungs is well adapted for the absorption of aerial poisons (gases and vapors), and conveying them at once over a large area into the blood. The action of the vapors of ether and chloroform, as well as of the poisonous gases—sulphuretted hydrogen and carbonic acid—furnish instances of the rapidity and energy with which these poisons produce their effects through the lungs.

Liquids which may be taken into the stomach in large doses without destroying life operate by their vapors in small quantity and with fatal effect through the pulmonary membrane. This is well exemplified in the use of chloroform vapor. In April, 1871, a healthy man, æt. 48, whose lungs and heart were sound, took chloroform vapor prior to a surgical operation for fistula. One drachm was given on a small square of lint. It produced insensibility in a few minutes, without causing any unusual symptoms to indicate danger. Suddenly the pulse stopped and the heart ceased to beat. The man was dead. If the deceased had swallowed this quantity of chloroform, it may be fairly inferred that it would have done him no injury. Adults have recovered from two, three, and four ounces of liquid chloroform taken into the stomach. The cause of the difference will be at once apparent. A small quantity, in the form of *vapor*, readily traverses the mucous membrane of the lungs and penetrates at once into the blood over a large area. In the form of a *liquid*, not very soluble in the fluids of the stomach, it is not rapidly absorbed by the gastric mucous membrane, and finds its way only slowly into the blood. The mode of administration by the lungs prevents elimination; but the liquid chloroform absorbed by the gastric membrane, may be, and probably is, eliminated by the lungs. Even when the quantity of gaseous poison is small, as where only a hundredth part of sulphuretted hydrogen or a tenth part of carbonic acid gas is diffused through the air, the noxious substance readily finds its way into the blood; and, being absorbed and circulated with greater rapidity than it is eliminated, the blood is permanently poisoned and death is the result. The frequency with which respiration is performed compensates for the small proportion of the poison diffused through the air. It is through this medium that poisonous miasmata and the poisons of contagious diseases are received into the body. There is reason to believe that even mineral substances, such as those of mercury, carbonate of lead, arsenious acid, and arsenite of copper in the state of impalpable dust, occasionally enter the blood through the lungs, and produce the usual effects of chronic poisoning. Horses, dogs, and rats have thus been poisoned in white-lead factories, in which the white lead was ground in a dry state, and therefore diffused as a fine dust in the air of the factory.

4. *Absorption by the Stomach.*—In all ordinary cases of poisoning, involving medico-legal inquiry, the poison enters the stomach and passes thence into the intestines. Thus the mucous membrane of these organs is the medium by which the substance is absorbed and conveyed into the blood. Absorption by the *stomach* is modified by the full or

empty condition of the organ. The process takes place more rapidly when the stomach is empty or the person is fasting. The acid secretions of the stomach increase the solubility of many substances, and promote their absorption. Thus nux vomica, in coarse powder, yields strychnia, and the sliced roots of aconite and œnanthe yield a sufficient quantity of their alkaloids to produce the ordinary symptoms with great rapidity, leaving the root or powder apparently unchanged in the stomach. Mineral poisons not dissolved by water, such as white precipitate, are rendered sufficiently soluble under the circumstances, to be absorbed and diffused by the blood.

It is a remarkable fact, that some poisons which operate fatally through a wound, or by injection into the blood, resist the absorbing action of the stomach in some cases entirely, in others partially. Bernard found that curara destroyed the life of a bird in a few seconds when a small quantity of it was injected into a wound, but when thrown into the stomach of a rabbit it had no effect, although on being removed from the stomach, its poisonous properties were unchanged, and it was still capable of causing death by injection into a wound. (*Op. cit.*, pp. 61, 239.) It was found, however, that if the animal was in a fasting state, the poison was sufficiently absorbed by the mucous membrane of the stomach to cause death. (*Op. cit.*, p. 291.) From this result, it is obvious, that under certain conditions, a poison which acts chiefly by a wound, may be still absorbed in fatal proportion by the mucous membrane of the stomach.

The poison of venomous serpents and the virus of glanders have presented similar anomalous results. One experimentalist observed that they acted through the stomach, while another found that they were inert when placed within this organ (*Med. Times and Gaz.*, April 11, 1857, p. 364). May not these conflicting results have depended on the comparative rapidity of absorption and elimination? When injected into a wound, the poison is carried into the blood more rapidly than it is eliminated by the kidneys or other organs. When taken into the stomach, it may be excreted by the urine and other fluids as rapidly as it is absorbed through the mucous membrane. In the case of an animal fasting, absorption would go on to a greater extent than elimination. The poison would thus accumulate in the blood and produce its usual effects.

In June, 1873, Dr. Pavy and I performed two experiments with the dry poison of the cobra de capello, which showed that even in a fasting animal, no effects were produced when it was injected into the stomach. Two grains of dry cobra poison were mixed with a small quantity of distilled water and introduced into a wound in the cellular tissue beneath the skin of a rabbit. Symptoms of poisoning showed themselves in a quarter of an hour, and the animal died in twenty minutes afterwards. A similar quantity of the cobra poison was injected into the stomach of a healthy young dog which had been kept without food for many hours. No symptoms of poisoning were at any time observed, and on the following day the dog was as well as usual, and took his food with appetite. (*Guy's Hosp. Reports*, 1874, p. 297.)

Dr. Fayrer found that the poison of the cobra recently emitted, was absorbed although slowly through serous and mucous membranes, and that when introduced into the stomach, put into the eye, or applied to the peritoneum, it might cause the death of animals, although not with the same certainty as when injected into a wound. (*Thanatophidia of India.*)

The Intestines.—The mucous surface of the small intestines absorbs poison with greater rapidity and uniformity than that of the stomach; and from experiments on dogs performed by Roselli and Gaetano Strombio, the same difference is observed with respect to the *rectum.* As arsenic, corrosive sublimate, laudanum, and other poisons have of late years been criminally administered in several instances in the form of enemata, these results are of some interest. A quarter of a grain of strychnia dissolved in spirit was administered to dogs, in one set of cases by the stomach, and in another by the rectum, care being taken that both were empty before the poison was introduced. The maximum period for the commencement of the symptoms by the *stomach* was in from thirteen to fifteen minutes, the minimum period from ten to twelve minutes; while by the *rectum* the periods were respectively ten to twelve minutes and four to ten minutes. The period of death also differed: the dog which received the poison by the stomach died in sixty-five minutes, while that which had the poison by the rectum died in forty minutes. It was further noticed that while the sixteenth part of a grain of strychnia killed three dogs when administered by the rectum—the tetanic spasms in two being very slight—the same dose of this poison given by the stomach did not cause death. In respect to the salts of morphia, the symptoms by the stomach commenced in from three to nine minutes, whereas by the rectum they appeared in from two to six minutes. (*Galtier, op. cit.,* 9.) Mr. Savory, of St. Bartholomew's, informs me that his experiments bear out the correctness of these results. While these facts possess a certain value in reference to the action of some poisons on the stomach and rectum, it is necessary to bear in mind that the period of commencement of symptoms and the period of death are variable in man and animals.

These results are of practical importance in relation to the use of powerful medicines applied to the rectum in the form of suppositories, or of injections. A quantity of opium, which might have been taken into the stomach without producing more than the ordinary medicinal effects, has caused profound narcotism by the rectum, and I have known an instance in which an injection of belladonna nearly destroyed life through its rapid absorption and powerful action by this portion of the intestines.

The mucous membrane of the eye (the conjunctiva) also absorbs poisons. This is well seen by the rapid effect of a solution of atropia, in causing a dilatation of the pupil. Dr. Fayrer found that when the cobra poison was applied to the mucous membrane of the eye, the effects were less dangerous than when it was injected into the blood. When placed on the conjunctiva of a dog, it caused symptoms of poisoning rapidly and strongly, although these were not in all cases fatally developed. One of his assistants had a very narrow escape, owing to a small portion of the poison falling on the conjunctiva of his eye, during an experiment. It was found, under these circumstances, to have an irritant action, for it produced violent inflammation of this membrane.

In his experiments with the extract of Calabar bean, Dr. Fraser observed that death took place most rapidly when the poison was injected into the circulation, or placed in contact with a wounded surface. It followed nearly as quickly when injected into a serous cavity, but more slowly when in contact with the mucous membrane of the stomach. Rabbits were killed by its application to the mucous membrane of the nose or eye. (*On the Calabar Bean*, p. 69.)

Some experiments have shown that direct contact of the poison with the mucous surface, or a wound, is not absolutely necessary to the process of absorption. Imbibition and percolation through porous substances will equally allow of the penetration of the poison into the blood, although the effects may be more slowly manifested. Mr. Horsley, of Cheltenham, gave to a dog two grains of strychnia in a pill with conserve of roses wrapped in thin paper. Three hours elapsed without any symptom of poisoning showing itself: in the morning the dog was found dead. When the stomach was opened the pill was found still inclosed in the paper wrapper, and on drying it, it was found to have lost only three-quarters of a grain. This had been removed by imbibition and absorption through the pores of the paper. Mr. Devonshire gave a grain of strychnia closely wrapped in paper to a cat. The animal died with the usual symptoms. The greater portion of the strychnia was found in the paper wrapper lying in the stomach of the cat. (See *Guy's Hospital Reports*, October, 1856, p. 336.) It must be obvious from these results that that portion of the poison only which passes into the body by absorption destroys life, and that in reference to strychnia, the quantity is very small. The portion which remains in the stomach unabsorbed has no share in causing the symptoms, or death; it is merely the surplus of the fatal dose, whether it be wrapped in paper, or lying on the surface of the stomach in a free state.

Hence it follows, that however large the quantity of poison taken, only a certain portion of it undergoes absorption: this constitutes the fatal dose, and it varies for each substance. Orfila found in his experiments on dogs, that on placing in a wound a certain weight of arsenic contained in a linen bag, the animal died when from one grain to two grains had been absorbed—the residue being retained in the bag. This is confirmatory of the results obtained by Mr. Horsley, from strychnia. In the experiment mentioned above, Mr. Horsley found that the dog died in six hours from the absorption of three-quarters of a grain of this alkaloid, showing that the poison had been removed from the paper bag in which it was inclosed, at the average rate of one-eighth of a grain in an hour.

In an experiment which I performed on a rabbit, with strychnia, the following results were obtained: One-eighth of a grain of acetate of strychnia, in powder, was placed in a wound beneath the skin of the animal. Symptoms of poisoning appeared in nine minutes, and the animal died in twenty minutes. On collecting and drying what remained in the wound, it was found to be about one-half of the quantity used, so that the animal had been killed by the absorption and diffusion of the sixteenth part of a grain of strychnia in twenty minutes. There was no doubt that the poison had been diffused by the blood through the whole of the body. Absorption had here gone on at the rate of one-fifth of a grain in an hour, the animal dying when the blood became over saturated. Had only one-sixteenth of a grain been used in the experiment, it is obvious that none would have remained—the whole would have been diffused with the blood throughout the body. Strychnia was, of course, easily detected in the wound in an unabsorbed state, but the most careful examination failed to show that there was any trace of the alkaloid in the liver, heart, or blood. The quantity deposited from such a small fractional dose was probably too small for detection.

CHAPTER IV.

DETECTION OF POISONS IN THE BLOOD BY THEIR CHEMICAL AND PHYSICAL PROPERTIES—BY SPECTRAL ANALYSIS—LOCAL ACTION OF POISONS—ELIMINATION AND DEPOSITION OF ABSORBED POISONS—CHANNELS OF EXIT—ELIMINATION OF ARSENIC AND OTHER POISONS BY THE URINE.

Detection of Poisons in the Blood.—That a large number of substances comprising medicines and poisons enter into the blood and are thereby diffused over the whole body, has been clearly established by the discovery of them in this liquid, as well as in the secretions and excretions derived from it, and in the soft organs, such as the liver, spleen, heart, and muscular system. This diffusion of mineral substances by means of the circulation was in the first instance established by experiments on animals. In the year 1832, the late Mr. Aston Key injected a solution of ferrocyanide of potassium into a wound on the inside of the thigh of a donkey. In six hours afterwards the animal was killed, and he forwarded to me for analysis a portion of blood taken from the femoral vein, another portion from the mesenteric veins, and lastly the contents of the thoracic ducts. The ferrocyanide was readily detected in the three specimens, being most abundant in the blood of the femoral vein, and least abundant in the contents of the thoracic duct.

In a set of experiments performed by Rapp, of Tubingen, with carbazotic or picric acid, a neurotic poison having an intensely colorific power, the fact of the universal diffusion of this substance through the blood was made evident after death by the *yellow color* imparted by it to the various textures and fluids of the body. In a fox killed in an hour and a half by swallowing sixteen grains of the acid, the conjunctiva (the white membrane of the eyes), the aqueous humor, the capsule of the lens, the membranes of the arteries, and in many places the cellular tissue, had acquired a lemon-yellow tint. In other experiments the stomach was dyed yellow; there was a yellowness of the fibrin of the blood, and the urine was tinged yellow. Although this poison affects the brain and spinal cord, producing convulsions and insensibility, it is a remarkable fact that in no instance was there any yellow tint in these parts. (*Christison on Poisons*, 4th ed., 796.) Similar results as to the coloration of the organs by this acid were obtained by the late Dr. Calvert, of Manchester. He further ascertained that the whole of the acid was eliminated in a few days, chiefly by the urine, to which it gave a yellow tint. Dr. Farquharson found that santonin, a substance which becomes yellow on exposure to light, imparted a strong saffron yellow color to the urine. A still more remarkable effect is that it dyes or stains the retina. Twenty minutes after swallowing five grains, he found that the flames of burning substances appeared of an intensely yellow color. In persons poisoned by sulphate of indigo, the urine, after the lapse of some hours, has been observed to acquire a blue color. This elimination of coloring matter by the urine is observed not only with respect to picric acid and indigo, but also in the red colors derived from anilin. M. Charnet injected a small quantity of the red dye fuchsin in solution beneath the skin of a guinea-pig, and it was observed that the urine passed by the animal soon after the injection was tinged of a deep red color. (*Ann. d'Hyg.*, 1863, vol. 2, p. 305.)

In some instances the *odor* of the poison clearly proves its diffusion. Prussic acid, the oil of bitter almonds, nitro-benzole, carbolic acid, camphor, alcohol, chloroform, ether, oil of turpentine, and, among the deadly agents, conia and nicotina, have been perceived by their odors, not only in the stomach, but in the brain and other parts of the body, to which they must have been conveyed by the blood. In poisoning with phosphorus, the diffusion of the poison is traceable not only by its garlic odor, but by its property of luminosity under slow oxidation. Thus, in the early stage of poisoning with this substance, the absorption and elimination are so rapid that the urine first passed by the patient is luminous in the dark. The intestines and even the flesh of animals poisoned with phosphorus have been observed to emit the odor of garlic, and in the dark they have appeared luminous. In the case of a woman who died while taking phosphorus medicinally, the whole of the viscera of the body were *luminous* in the dark. Physical facts of this description, by which the presence of poison is rendered evident to the senses, show even in a more satisfactory manner than chemical tests, that the soft parts of the body are universally penetrated by the substance absorbed.

In cases in which neither color nor odor will aid the inquirer, chemistry serves to reveal the presence of the poison and the extent of its distribution. The important discovery first announced by Orfila in 1839, that arsenic could be detected and separated from the blood, secretions, or viscera of persons who had died from its effects, produced a complete revolution in this department of toxicology. It mattered not from what part of the body the blood was taken, arsenic was equally discovered; so that, from these and other experiments, it appeared that the living or dead body in a case of arsenical poisoning, was for a time penetrated throughout by the poison, and that during life it was eliminated by the urine and other secretions. The fact that arsenic may be detected in the urine of a person who survives its effects, is a point of considerable importance in a medico-legal view. Thus an analysis of this liquid may furnish evidence otherwise only satisfactorily obtained by an examination of the dead body; and cases of the criminal administration of arsenic to the living, which had hitherto escaped the hands of justice, owing to the absence of chemical proofs, have thus become as clearly established to the satisfaction of a jury, as if the poison had operated fatally and had been found after death in the stomach. In all doubtful cases of poisoning, the detection of the poison in the urine is a great aid to diagnosis.

These remarks upon absorption and diffusion equally apply to the *alkaloidal* poisons. In a few instances these may be separated from the tissues by chemical processes, while, in others, their diffusion by absorption is indicated by their physiological effects. Thus atropia, the poison of belladonna, daturia, of stramonium, and hyoscyamia, of henbane, cause a dilatation of the pupil of the eye (mydriasis), while morphia, the poison of opium, and physostigmia, the poison of the Calabar bean, indicate their presence in the circulation among other symptoms by a contraction of the pupils. Some of these alkaloids in minute quantity impart to organic liquids and extracts, an extreme bitterness of *taste: e. g.*, strychnia, the poison of nux vomica, and picrotoxia, the poison of cocculus indicus. Others are attended with a powerful odor: *e. g.*, prussic acid, nicotina, the poison of tobacco, and conia, the poison of hemlock. The flesh and milk of animals fed on wormwood acquire a bitter taste from absorption of the bitter principle. Milk rendered bitter by it has proved noxious to an infant. (*Pereira*, vol. 2, pt. 2, p. 23.) Modern discoveries have led to the detection of substances in the body of such minute quantities as to be beyond the reach of the most delicate chemical processes. The salts of thallium exert a poisonous action on animals. The late Dr. Bence Jones found that although this metal could not be detected in an absorbed state in the tissues, by any chemical process, it was readily detected in the liver of a rabbit thus poisoned, by the aid of the spectroscope. The vivid bright green band characteristic of thallium, appeared in the spectrum from the burning of a small fragment of the dried liver of the animal. The salts of lithium have also been thus detected in the tissues by the bright red band in the spectrum characteristic of this metal. Spectral or optical analysis has, therefore, confirmed the conclusions of the earlier toxicologists regarding the absorption and universal diffusion of poisons in the body. This mode of analysis is, however, at present very limited in its range.

These facts afford all the evidence that need be desired to prove that substances acting as poisons, enter into, and are diffused through the body by the blood. With respect to those which have not yet been detected in an absorbed state, it may be reasonably inferred that this is simply owing to the imperfection of our methods of research.

There are some substances which owe their lethal action to the local effects which they produce on the living tissues, and although they may enter the blood by absorption, this entrance does not appear to be necessary to their noxious operation. The nitrate of silver is a corrosive poison, and proves fatal by producing an extensive destruction and disorganization of the viscera. Absorption does not appear to be necessary to its fatal action, yet it is undoubted that when this substance is given in small doses for medicinal purposes, it is conveyed in some form into the circulation—a fact established by the peculiar discoloration of the skin of the face and hands, produced by its long-continued employment in medicinal doses. The mineral acids appear to act chiefly by the local effects which they produce. Some have thought that they acted by absorption. One of them, nitric acid, has destroyed life by its vapor when received into the lungs. The symptoms and appearances were such as would arise from local injury done to the lungs. They were in no way connected with the absorption of the acid into the blood.

The arrow-poison of the Bari tribe of Central Africa appears to destroy life by a local action. Sir Samuel Baker describes it as a milky juice from a species of euphorbia. It is smeared upon the barbed blade of the arrow and dries into a resinous layer. It causes severe inflammation in the part, with sloughing and destruction of the surrounding muscles. It does not appear to act by absorption, for he saw a man who had been thus wounded in the leg five months before. The entire foot had sloughed away, leaving the bone exposed above the ankle. (*Albert N'Yanza*, vol. 1, p. 87.) This arrow-poison may be regarded as a variety of euphorbium, which has been long known for its acrid, irritating properties. Its action is generally confined to the parts which it touches, producing severe pain and inflammation, with vesication and sloughing. According to Wibmer (*Arzneimittel und Gifte*, art. Euphorbia), it is not absorbed, but its lethal action is confined to the parts with which it comes in contact.

Elimination and Deposition of Poisons.—It is now generally admitted by toxicologists that poisons which have been carried into the blood by absorption through the skin or mucous membrane, are diffused throughout the body. A portion is eliminated by the secretions, while another portion is deposited in the tissues; but elimination commences soon after the poison is absorbed, and continues so long as the person survives its effects. All the secretions share in this process of elimination. Thus organic and inorganic poisons have been found in the urine, bile, milk, saliva, serum of the serous membranes, and in the mucous secretions of the mucous membranes. Of all these liquids, the *urine* appears to be the principal and most constant channel; but there is reason to believe that some poisons pass more rapidly by certain secretions than others. Most of these fluids admit of special examination, excepting the milk, which of course can only present itself casually as a medium. The urine is in almost all cases accessible, and this gives the most satisfactory chemical results.

That substances, whether regarded as poisons or medicines, are rapidly removed from the body after absorption, is a fact now well known to physiologists. This is especially observed with respect to those bodies which have no chemical action on the blood, or which do not form insoluble compounds with the tissues. The iodide of potassium taken into the stomach has been found in the urine in less than *ten minutes*. Mr. Erichsen ascertained that the ferrocyanide of potassium might be detected in the urine two minutes after the administration of forty grains of the salt, and that the elimination was complete in twenty-four hours. (*Med. Gaz.*, vol. 36, pp. 363–410.)

Experiments on animals show that within one hour and a half arsenic may be extensively diffused throughout the body, and that elimination commences within this period. Orfila quotes experiments in which arsenic was found in the urine of horses (passed while the animal was living) in from three hours and a half to seven hours after the administration of the poison. But it was present in the urine contained in the bladder of a dead horse, within the short period of an hour after its administration. In dogs it was detected in the urine passed in from three to five hours after the injection of the arsenic. (*Toxicologie*, 1852, vol. 1, pp. 381–383.)

There are but few observations respecting the earliest time at which the poison shows itself in the urine of a person laboring under its effects; but there is reason to believe that it may be detected within three or four hours. In the case of a child, which fell under the observation of the late Dr. Geoghegan, no arsenic could be discovered in the small quantity of urine passed up to the fourteenth hour, and that passed from fourteen to thirty-six hours yielded only faint indications. It generally continues to be discharged with the urine so long as a person lives, but gradually diminishing in quantity. Orfila inferred from his observations that all the absorbed arsenic might be carried out of the body and cease to appear in the urine in from twelve to fifteen days; but in one case Dr. Geoghegan examined six ounces of urine passed on the sixth day, and could detect therein no trace of arsenic. There is some uncertainty regarding its presence in this secretion. It does not always appear to be the channel of elimination, and in cases of arsenical poisoning, the secretion of urine is sometimes greatly diminished or wholly suppressed. In the case of the Duke de Praslin, who died six days after he had taken a large dose of arsenic, the poison was found in the liver and in the intestines, but none was found in ten ounces of the urine passed shortly before his death. (*Ann. d'Hyg.*, 1847, p. 402.) In giving arsenic to dogs for a period of nine months, in doses gradually increased, Danger and Flandin state that they repeatedly analyzed the urine without finding arsenic. (*Toxicologie*, vol. 1, p. 737.)

A case of poisoning with arsenic occurred to Dr. Maclagan, of Edinburgh, in which the gradual disappearance of the poison from the urine was traced over a long period. A woman swallowed half a dessertspoonful of arsenic on November 4, and was under treatment until the 29th, when she left the hospital, having recovered from the effects of the poison. There was, in the first instance, active vomiting; and on analysis it was found that the vomited matters contained arsenic. The urine was not examined until the second day (November 6). It then gave, by Marsh's process, an abundant arsenical deposit. On the fourth day, twelve ounces also gave a copious deposit of arsenic. On the fifth day, ten ounces gave a smaller quantity. On the ninth day, the poison was still found; on the fifteenth day, twenty-four ounces gave only a small quantity; on the twenty-first day, twenty-six ounces gave a faint deposit; and on the twenty-fifth day, not a trace could be detected. (*Ed. Monthly Journal*, vol. 14, p. 131, 1852.)

In the case of a boy, who was a patient under Dr. Wilks, at Guy's Hospital, arsenic was given daily in the proportion of one-eighth of a grain, divided into three doses, for a period of seventy days consecutively. The quantity taken altogether amounted to nine grains, sufficient to destroy four adults in the absence of elimination. The arsenic was discontinued, and the urine was examined daily for *ten days*. It was found in from four to six ounces of urine, but in decreasing quantity. At the end of this time the boy ran away, so that it was impossible to carry the experiments further.

These facts lead to the conclusion that although the urine is an important channel of elimination, we cannot place absolute reliance on a negative result. The detection of it in this secretion shows that the poison has been taken; but the non-detection of it does not necessarily show that there is no arsenic in the body.

If a poison is eliminated by the urine or other fluids as rapidly as it is carried into the blood by absorption, no symptoms of poisoning are produced. If it is more rapidly absorbed than eliminated, it will accumulate in the blood and operate with fatal effect. It is, therefore, not so much the quantity taken as the quantity absorbed in a given time, which determines the lethal operation of these agents. This quantity may be so small that, although it may produce symptoms, it will not destroy life. This is occasionally witnessed in medical practice, where medicinal doses of arsenic, strychnia, or prussic acid have been slightly exceeded. It is at this point that we reach the boundary which separates a poison from a medicine.

It will be understood, therefore, that the accumulation in the blood of a certain quantity of poison is required before symptoms show themselves, and the rate of blood-saturation varies according to circumstances. The occurrence of certain symptoms may be taken as the indication of the poison having reached the blood, and produced its effects on that liquid. Thus faintness, syncope, and general depression, with an indescribable uneasiness are among the first symptoms caused by absorbed arsenic, and in a series of cases which I was required to examine, these symptoms showed themselves in from five to ten minutes after the poison had been taken in a state of solution. Absorbed morphia is indicated by drowsiness, stupor, and an irresistible tendency to sleep—and strychnia by shivering and shuddering, with convulsive twitchings of the muscles.

Saline substances given medicinally may be taken in large quantities and passed through the body without doing injury, provided the doses are not too large, or follow each other at too short an interval. Nitre is safely used as a diuretic, but in a dose of one ounce, taken at once, it has operated as an irritant poison, and caused death in three hours. A patient in Guy's Hospital, under Dr. Wilks, took in forty-six days twenty-five ounces and six drachms of this compound. The doses were increased from half a drachm to one drachm and a half thrice daily. The nitre was eliminated by the urine in the proportion of 2.48 grains to an ounce, representing in the quantity of urine passed in twenty-four hours, 158.7 grains of nitre, at a time when the patient was taking daily 270 grains of the salt in three doses. (*Guy's Hosp. Reports*, 1863, p. 173.) The remainder no doubt passed off by the intestines like other saline medicines. Thus in a period of less than seven weeks, this man had swallowed, not only with impunity, but with benefit, a quantity of nitre sufficient to kill twenty-five adults.

Arsenic, strychnia, and all medicines capable of acting as poisons follow the same rule. If a sufficient interval be allowed for elimination, they may be prescribed in quantities which, if taken at once, would speedily destroy life. (*Dr. Wilks's case*, p. 24.) Mr. T. Turner, who had had a large Indian experience in the medicinal use of arsenic in intermittent fevers, gave to a recruit, æt. 22, as much as *nineteen grains* of arsenic, or nine fatal doses, in twenty-eight days. The man took a grain and a half within ten hours without any gastric disturbance. (*Med. Times and Gaz.*, Sept. 28, 1861, p. 315.) There is no doubt that there was rapid elimination, and probably the disease gave a certain degree of tolerance.

As a rule, therefore, poisons which are absorbed do not accumulate in the body unless elimination is arrested or retarded.

CHAPTER V.

ABSORPTION, ELIMINATION, AND DEPOSITION OF POISONS—ILLUSTRATED BY CASES OF ARSENICAL POISONING—APPEARANCE OF POISON IN THE URINE—PERIOD FOR COMPLETE ELIMINATION OF ABSORBED POISON—MEDICO-LEGAL QUESTIONS—DATE OF ADMINISTRATION—DETECTION IN THE LIVER AND OTHER ORGANS—ALLEGED PRESENCE IN THE HAIR—ELIMINATION OF ANTIMONY.

WHILE one portion of an absorbed poison is undergoing elimination by the urine and other secretions, another is temporarily deposited in the liver and other viscera. According to M. Flandin the largest proportion will be found in the liver, and after this organ in the kidneys. From the observations made by the late Dr. Geoghegan it would appear that the deposit of arsenic in the liver continues to increase up to about fifteen hours after the poison has been taken. It then gradually diminishes, and if the person should survive, it entirely disappears in from fourteen to seventeen days. The various cases which were examined by Dr. Geoghegan yielded the following results. Assuming the average weight of the human liver to be three and a half pounds, the total amount of arsenic deposited in this organ was:

After taking the poison.	Total weight of arsenic.
In 5½ to 7 hours,	0.8 grains.
8¾ "	1.2 "
15 "	2.0 "
17 to 20 "	1.3 "
10½ days,	1.5 "
14 days,	0.17 "
17 days,	nil.

Arsenic has destroyed life in *two hours*, but the liver has not been examined in these cases, which are quite exceptional. I have found arsenic in the liver in *four* hours, and in another instance in six hours after the taking of the poison; but these are the earliest periods. In the greater proportion of cases persons survive the effects of this poison for ten hours. At this period it is found not only in the liver, but in the other soft organs. In a case proving fatal in ten hours M. Chevallier detected the poison in the liver, as well as in the stomach, intestines, and spleen. (*Ann. d'Hyg.*, 1848, vol. 1, p. 419.) It appears to be eliminated from the liver by the bile, in which liquid it is frequently found. Although Dr. Geoghegan's observations lead to the conclusion that the liver acquires its maximum saturation in fifteen hours, it is improbable, as he remarks, that such quantitative results should be the same in all cases.

Dr. Geoghegan's results corroborate the observations of Orfila regarding the disappearance of arsenic from the liver, namely, that at or about the fourteenth day from the date of the poisoning, absorbed arsenic has either disappeared or is rapidly disappearing from this organ. It may not be found at an earlier date. Thus, in one protracted case of poisoning with arsenic, which proved fatal after *seven* days, I could detect no arsenic in the liver. (*Guy's Hosp. Rep.*, vol. 7, p. 194.) It may, however, usually be found up to the fourteenth day if the person survives so long. In a clear case of poisoning with arsenic the late Mr. Herapath could detect no trace of arsenic in the body of a person who died in the fifteenth day after taking the poison. (*The Queen* v. *Williams*, South Wales Circuit, July, 1863.) This chemical expert stated in reference to this result that neither in his reading nor in his experience had he known arsenic to have been detected fifteen days after its administration. (*Lancet*, July 11, 1863, p. 47.) This statement is borne out by the case of Dr. Alexander, of which the particulars were communicated to me by Dr. Geoghegan. On March 16, 1857, this gentleman took unknowingly a quantity of arsenic in arrowroot. It had been mixed with the arrowroot by mistake. The usual symptoms followed, and he died on April 1. The appearances were such as arsenic would produce; the stomach was ulcerated. Dr. Geoghegan made an analysis of the stomach and its contents, of the liver, spleen, and other viscera; but there was no arsenic in any of the organs, although the poison was abundantly contained in the arrowroot eaten by deceased. Thus, in *seventeen days*, the arsenic had been completely eliminated. This case further shows distinctly, in opposition to the rash assertions of some medical witnesses, claiming to speak with authority, that a person may die from poison, and yet no trace of one of the most easily detectable poisons, will be found in the body after death! (See *Med. Times and Gazette*, April 18, 1857, p. 388.)

From these observations it is reasonable to infer that when a person survives from fifteen to twenty-one days, absorbed arsenic will not be found in the soft organs. Under treatment it may entirely disappear in a few days from the contents of the stomach and bowels. In a case of this kind, the question may arise—if no poison is present in the body—Of what does the person die? Death in such cases may take place from exhaustion, or the effects of the poison on the brain and nervous system. In Mr. Herapath's case, as well as in that recorded by Dr. Geoghegan, the patients died from exhaustion. It is not absolutely necessary that some of the poison should remain in the body at the time of death, in order to account for death under these circumstances.

The facts connected with the elimination and deposition in the tissues, of arsenic and other poisons, have given rise to important questions respecting the date of administration, and the discovery or non-discovery of these agents by chemical processes in the fluids and solids of the body. Within what time, after administration, will a poison be deposited in the tissues? How long will a poison once deposited in an organ, remain there? By what channel is it eliminated? When does this elimination commence, and when is it completed? The facts obtained from the human body and from experiments on animals show great differences among the different poisons. The functions of absorption and elimination are probably not the same in man and animals; and among human beings it may be considered that they are performed more rapidly in the child than in the adult, in the female than in the male, and in the healthy and vigorous than in aged persons. The condition of the body must also affect these functions; and the effects produced by the poison itself must, to a certain extent, influence them.

In *Reg.* v. *Hunter* (Liverpool, Lent Ass., 1843), the prisoner was acquitted of the charge of poisoning, chiefly on the ground that no arsenic was detected in the body, although the deceased had died in three days after the alleged administration of the poison.

Although no definite conclusions can be drawn regarding the period and relative amount of deposit in the soft organs, or the period at which absorbed arsenic is entirely discharged from the body, there are certain leading points which are undisputed. In the first place, arsenic is not a normal constituent of the human body; this has been clearly proved by Orfila, under the eyes of a committee of scientific men. (*Rapport sur les Moyens de constater la présence de l'Arsénic dans les Empoisonnements par ce Toxique*, par M. Orfila, Paris, 1841.) Secondly, when introduced as a poison, it is absorbed, and although temporarily deposited in some of the organs, it is sooner or later eliminated, and the whole is removed from the body. The statement that when once deposited it may remain for an indefinite period, has no foundation in fact. In recent cases of administration it may be found in the stomach and bowels, and not in the liver or other organs, while in cases of older date it may be found in the liver, after it has disappeared entirely from the stomach. Thus, in the cases of the Atlee family, referred to me by Mr. Carter, coroner for Surrey, in January, 1854, the body of the woman was exhumed after a month's burial. Arsenic was not found in the *stomach* or *bowels*, but it was readily detected in a small portion of the *liver*. The poison had probably been taken several days before death.

The kidneys, spleen, heart, lungs, and brain, and after these organs the muscles and bones, are also the seats of deposit, and the proportion deposited, so far as it is yet known, is in the order in which these parts are mentioned. The analysis for absorbed poison rarely extends beyond the liver and kidneys, for if not found in these organs, it is not likely to be found in the other organs mentioned.

Arsenic has been found deposited in the bones, but in very small quantity. In one case, a medico-legal question arose whether it was ever deposited in the *hair*. A wealthy old lady died after a protracted illness ending in dropsy. A female relative, disappointed with the terms of her will, asserted that she had been murdered by the administration of small doses of arsenic over a long period. The woman went before the authorities, and stated that she had cut off some of the deceased's hair while the body was lying in the coffin, that the hair had been analyzed by a chemist, and arsenic found it it. The case was subsequently submitted to Dr. Casper and Prof. Mitscherlich, of Berlin, with the result that, on an accurate analysis of a portion of hair really cut from the head of deceased, not a trace of arsenic was found in it. The story told by the woman, who brought the charge of poisoning, was altogether improbable. There was no proof that the hair which she caused to be analyzed was taken from the head of deceased, and she made no application for an analysis until after she had read in some medical book that the hair of animals, poisoned by arsenic, contained that mineral. There was also good reason to suspect that she had tampered with the hair. On the result of Mitscherlich's analysis, the complaint was dismissed as unfounded. (Casper, *Gericht. Med.*, vol. 1, p. 419, 1857.) There is no record of arsenic ever having been discovered in the hair of persons poisoned by this substance. I have examined the feathers of birds poisoned with arsenic, but none of the poison could be detected in them, although it was found in the bones, beak, and claws.

With reference to absorbed arsenic, there are two points requiring notice: 1. The extent to which it is diffused through the body, and 2. The absolute quantity deposited in the organs. In chronic poisoning, arising from the administration of small doses at intervals, I have found the arsenic extensively diffused, but in small proportion. The quantity deposited appears to depend on the largeness of the dose, or on the frequency with which small doses are repeated. There are some facts on record which show to how great an extent arsenic may be diffused when it has once entered the blood. In the case of a pregnant woman poisoned by arsenic in the fourth month of pregnancy, the poison was detected, by MM. Mareska and Lardos, in the body of the fœtus. It was also discovered in the uterus and placenta, the latter organ containing a larger proportion than the fœtus, but there was none in the liquor amnii. (*Gaz. des Hôpitaux*, Janvier, 1846.) Even the entozoa found in the human body became under these circumstances thoroughly impregnated with the poison. (*Guy's Hosp. Rep.*, October, 1846, p. 462.) In March, 1857, some fowls which had died from the effects of arsenic were submitted to examination. In the crop and gizzard of a fine cock about twenty grains of the poison were found, and the whole body was thoroughly saturated with absorbed arsenic. The poison was separated from the blood, the liver, the muscles of the breast, the comb, the claws, and even the thigh-bones. In examining the body of a hen, the arsenic was found in large quantity in the ova contained in the oviduct, and particularly in the yolks of those which were developed.

These results are subject to exceptions, the causes of which are not well understood. Arsenic is not always found in the blood, and a much larger quantity of this liquid than of the liver is required for its detection. It is not necessarily present in all the organs. It may be found in the liver or kidneys, but not in the heart or muscles. In a case examined by the late Dr. Geoghegan, the liver yielded a small quantity of arsenic, but none could be extracted from the blood. Again, in a case fatal in nine days, he discovered no arsenic in the muscular structure; while in another case, fatal in seven hours, he discovered it without difficulty in the proportion of one-thirteenth of a grain to the pound. (*Op. cit.*, p. 113.)

The observations here made respecting the absorption and elimination of arsenic are more or less applicable to antimony and other metallic poisons. Antimony, like arsenic, may be detected in the urine, passed a few hours after its administration; and, from Orfila's experiments, there is no reason to believe that this metal would remain deposited in the soft organs longer than fifteen or twenty days after the last date of administration.

The presence of the poison (such as arsenic or antimony) in the stomach or bowels has generally been taken to indicate a recent administration by the mouth or rectum. If the poison be in some quantity, and in a solid or liquid form in the contents, this inference is justifiable, but if only in traces in the coats or in the mucus of the stomach, it may be the residue of a quantity taken by the mouth some days previously, or the result of an elimination by the mucous secretions of the stomach and intestines. When the poison is found in the stomach and intestines and not in the other viscera, and at the same time there has been no application to a wound or ulcer, it is reasonable to presume that its presence is the result of ingestion into these parts, and not of elimination from the mucous surface.

On the absorption, elimination, and deposition of mercury, lead, and copper, some remarks will be made in treating of the poisonous salts of these metals.

By the aid of spectral analysis Dr. Dupré was enabled to trace the complete elimination of the salts of lithium from the urine. It was found to be completed in five or six days. (*Proc. R. S.*, March, 1872, p. 268.)

CHAPTER VI.

ABSORPTION AND ELIMINATION OF ACID AND ALKALINE POISONS—OF LIQUID AND VOLATILE POISONS—RAPID DIFFUSION OF PRUSSIC ACID—ELIMINATION OF MORPHIA—STRYCHNIA, ITS DEPOSITION IN THE TISSUES—EXPERIMENTS ON ANIMALS—OBSERVATIONS ON MAN—ITS ENTIRE REMOVAL FROM THE BODY —PROOFS OF ABSORPTION OF OTHER ALKALOIDS.

Acid Poisons.—It has been a disputed question, whether *sulphuric acid* is or is not absorbed and carried into the circulation in cases of acute poisoning. M. Bouchardat considers that it is absorbed, and that it causes death by leading to a coagulation of the blood in the heart, aorta, and large bloodvessels. He has found these coagula in two cases in considerable quantity; and in one of them, the lining membrane of the aorta was reddened. (*Annales d'Hygiène,* 1837, vol. 1, p. 362.) I have observed this last-mentioned appearance in one case, as well as the occurrence of coagula in two instances; but there does not seem to be any reason for believing that they result from the action of a portion of absorbed sulphuric acid. (Galtier, *Toxicologie,* 1, 190, 191.) In analyzing these coagula taken from a person who had died from the effects of sulphuric acid, I did not find a trace of the acid in them. According to Orfila, the absorption of the mineral acids may take place owing to their compounds with albumen being soluble and neutral. There is no doubt that these albuminous compounds are soluble in a large quantity of water, but they are insoluble when much acid is present. In a case reported by Dr. Letheby to the Pathological Society, a chemical analysis of the urine led him to the inference that the acid was rapidly eliminated by this secretion. The quantity thus passed within four days was considerable. (*Med. Gaz.,* vol. 39, p. 116.)

The reader will find that this subject has been fully examined by the late Dr. Geoghegan. (*Med. Gazette,* vol. 48, p. 330.) In a case in which a woman swallowed one ounce and a half of oil of vitriol, and survived thirty-one hours, he states that he found traces of sulphuric acid in the serum of the pericardium and in the kidney. There was none in the blood, but a quantity of free phosphoric acid, which he considered to be the equivalent of the sulphuric acid which had been absorbed and had decomposed the alkaline phosphate of the blood, the alkaline sulphate produced having been eliminated by the kidney. The stomach was perforated, and the organ empty. In a case of poisoning with sulphuric acid, which occurred to Dr. Walker, we are informed that there was a trace of sulphuric acid in the serous fluid at the base of the brain, and a larger quantity in the blood contained in the heart. There was none in the stomach, and only a slight trace in the duodenum. (*Ed. Monthly Journ.,* June, 1850, p. 538.)

It will be apparent from these statements, that the results of experiments for the detection of absorbed sulphuric acid in the blood are not in accordance. According to Casper, in this form of poisoning the blood has always an acid reaction, even in the organs which are healthy; it has also a treacly consistency and a cherry-red color. He found, in one instance, the pericardial fluid acid, and in another, the case of a pregnant woman, the amniotic fluid was acid (*Handbuch der Ger. Medicin*, 1857, vol. 1, p. 400, 430); but he did not test the liquid for sulphuric or phosphoric acid. Professor Carus has reported a case in which sulphuric acid was taken by a pregnant woman, and it was found not only in the water of the amnios, but in the cavity of the pleura and of the peritoneum of the fœtus, as well as in the heart and bladder. (*Beck's Med. Jur.*, vol. 2, p. 429; and *Bulletin des Sciences Médicales*, vol. 13, p. 72.)

The acidity of the organs and flesh after death cannot alone be relied on as evidence of the presence of an acid poison in the blood, unless sulphuric acid is actually detected in those parts. It is now well known that the liver and some other organs, as well as the muscles, acquire an acid reaction as the result of spontaneous changes in the principles contained in them, taking place after death. If the acid has permeated through the coats of the stomach and intestines, and impregnated the organs around, the fact will be indicated by the application of chemical tests.

Orfila states that he separated *nitric acid* from the urine of animals poisoned by the diluted acid. He distilled the urine with sulphuric acid, neutralized by potash the acid liquid thus obtained, and succeeded in procuring nitrate of potash. The result was not uniformly the same. At certain stages of the poisoning only the urine was found to contain nitric acid (*Toxicologie*, 1852, vol. 1, p. 185). As the nitrates are not constituents of the urine, the fact may be of importance, although the circumstances under which nitric acid was thus procured are not likely to present themselves in a case of acute poisoning in a human being.

Oxalic acid is supposed to enter the blood, and give it a dark brown color. In a case which proved rapidly fatal, where two ounces of the poison had been taken, I examined four ounces of blood taken from the vena cava, but not a trace of oxalic acid could be detected. Dr. Christison states that he did not succeed in detecting the poison in the blood even when a solution of it had been purposely injected into the femoral vein of an animal which died in thirty seconds. Orfila was unable to obtain any traces of it from the livers or spleens of animals poisoned by the acid. (*Op. cit.*, 1, 247.) These negative results may be explained by supposing that the poison is decomposed, or the usual method of separating it from organic compounds is not sufficiently delicate. In two cases it is stated that leeches have been killed by the blood drawn by them from persons who were at the time laboring under the effects of this poison. This seems to render it probable that the blood is poisoned, and, indeed, Orfila states that he succeeded in detecting oxalic acid in the urine, although not in the solid organs. (*Toxicol.*, vol. 1, p. 190.) According to Wöhler, it may be detected under the form of oxalate of lime in the urine of animals to which it has been administered. This fact should not be lost sight of by the medical jurist, as the oxalate of lime, although frequently found in certain states of disease, is not a normal constituent of urine. The microscope would here render great assistance, as the octahedral form of the oxalate of lime is peculiar. It is probable that, in acute cases, death is solely to be ascribed to the absorption of the poison and its peculiar action on the blood.

According to the experiments of Orfila, *potash* is absorbed and conveyed into the blood. The alkali is eliminated by the urine, which is thereby rendered alkaline. When he gave about one drachm of potash to dogs, the presence of the alkali was detected after the lapse of six hours in the liver, spleen, and kidneys. Owing to the solvent action of this poison on fibrin and albumen, the blood, although it may be darker in color, is never found coagulated in the vessels after death.

Poisons of a liquid and volatile nature enter the blood, are diffused through the body, and eliminated with great rapidity. Prussic acid and alcohol furnish instances of this rapid absorption and elimination. These volatile poisons enable us to solve an important practical question which often presents itself, namely: Within what time after administration is the poison absorbed, so as to produce its usual effects? Müller found in his experiments that a liquid poison brought into contact with a wounded surface might be absorbed and distributed through the body in from half a minute to two minutes.

This result is fully explained by the great rapidity of the circulation. From the capacity of the cavity of the heart, and the number of contractions which take place in a minute, it is estimated that one hundred and forty ounces of blood pass through the heart within this short period of time. Thus all the blood in the adult body would pass through the heart in three minutes. According to Valentin, however, one minute would suffice. (*Kirke's Physiology*, p. 139.) Substances injected into one jugular vein have been detected in the blood of the opposite jugular, in from twenty to thirty seconds. Dr. Blake's experiments point to even a greater rapidity of distribution. He found that a solution of nitrate of barium injected into the jugular vein of a horse could be detected in blood drawn from the carotid artery of the opposite side in from fifteen to twenty seconds after the injection. In a dog, the poisonous effects of strychnia on the nervous system were manifested in twelve seconds after injection into the jugular vein; in a fowl, in six and a half seconds; and in a rabbit, in four and a half seconds. The general conclusion drawn by Blake was that a poison might be diffused through the human body in so short a period of time as *nine seconds;* and he states that an interval of more than nine seconds always elapsed between the introduction of a poison into the capillaries, or veins, and the appearance of the first symptoms.

In reference to most poisons which enter the body by the mouth, some minutes, or even hours, may pass before the appearance of symptoms. This will depend on the nature of the poison, its physical condition, the amount absorbed in a given time, and the quantity accumulated in the blood.

Prussic Acid.—Kramer discovered prussic acid in the blood of an animal which died in *thirty-six seconds* after its administration in the ordinary way. Mr. Waterworth, formerly a pupil at Guy's Hospital, gave to an animal a fatal dose of prussic acid, and in less than a *minute* afterwards, when all signs of life had ceased, he made an opening into the chest, and tested the warm vapor which escaped from it. In this vapor he clearly detected prussic acid. This appears to establish the correctness of Müller's view in respect to the very rapid diffusion of this poison. These volatile poisons, like the soluble gases, are chiefly eliminated in vapor by the lungs. So long as life remains, the peculiar odor of the poison is plainly perceptible in the breath. Although this poison may not be detected in the body either by its odor or vapor, when some weeks or months have elapsed, this does not prove that it was entirely eliminated at the time of death. In the presence of decomposing animal matter, containing sulphide of ammonium, a portion of it is converted into sulphocyanide of ammonium. (See Prussic Acid, *post.*)

The Alkaloids.—There can be no doubt that these powerful agents, of which morphia and strychnia may be taken as types, are absorbed into the blood and diffused through the system like other poisons. Accurate observations on the absorption and elimination of morphia in cases of poisoning in the human body are rare. The facts hitherto noticed tend to show that in a poisonous dose it is rapidly removed from the stomach by absorption, and that the residue of a fatal dose is seldom found when the person has survived some hours. In a case which occurred to me in March, 1863, a man æt. 52 died in *ten hours* from a dose of one grain of the hydrochlorate. No trace of morphia remained in the stomach. In another case communicated to me a man died in *thirteen hours* from a dose of one grain of the hydrochlorate taken in a pill; no morphia could be detected in the contents of the stomach. It is to be observed that this alkaloid is more difficult of separation from organic matter than strychnia, and when separated, the tests for its identification are not so conclusive. It has been found deposited in the tissues in some rare cases by competent analysts. In a case of poisoning, which occurred at Bruges in 1845, M. Stas states that he detected morphia in the viscera of a body after an interment of thirteen months; and in 1847 he detected and separated this alkaloid from the viscera of another body. This proves that morphia is absorbed and deposited, and also that it resists decomposition for a long time. The facts throw no light upon the rate of absorption or the date of entire elimination: for we have no information on the quantity of morphia taken by the deceased persons—the period which they survived, the quantity found in the viscera—and the viscera in which the poison was detected.

Strychnia.—In the year 1827, Vernière first showed by an ingenious physiological experiment that the poison of nux vomica (strychnia) entered the venous blood by absorption, and that the blood thus impregnated, when transfused into another animal, produced the usual symptoms of strychnia poisoning. It is probable that if a very large dose of nux vomica could be given to one animal, and, while laboring under its effects, a sufficient quantity of blood could be safely taken from it and transfused into the body of another, it might be found that this liquid would act as a poison and cause death. There are, however, insuperable obstacles to the performance of such an experiment;

because if a large dose of poison be given to the first animal, it may die before a sufficient quantity of blood is transfused from it. If a small quantity of poison be given, or a small quantity of blood be transfused, the poison might be so diluted by diffusion that no fair inference could be drawn from the results. On the other hand, if a large quantity of blood be transfused, this alone might cause the death of the animal which lost the blood, and yet not be sufficient to produce fatal effects in the other.

The rapidity with which strychnia is absorbed and diffused through the body varies probably according to many circumstances. On the fact of its diffusion, there is one set of experiments by Mr. Blake: he found, on introducing the nitrate of strychnia into a vein, that the action of the poison on the spinal cord was manifested by tetanic convulsions in sixteen seconds in the horse, in twelve seconds in the dog, in six and a half seconds in the fowl, and in four and a half seconds in the rabbit. Severe symptoms are not produced until the poison is diffused through the circulation; and the more rapidly it enters the blood, the more speedily do the effects appear. This shows that accumulation by absorption is chiefly concerned in the operation of this poison. Sir R. Christison killed a dog in *two minutes*, with the sixth part of a grain dissolved in alcohol, injected into the chest; and a wild boar was killed in *ten minutes* with one-third of a grain. An instance has been privately communicated to me in which a man died in *ten minutes* from a dose of ten grains in solution! This is the most rapid case of death yet known; and there must have been here very speedy absorption and diffusion. Dr. Harley injected one-twelfth of a grain of acetate of strychnia in solution into the jugular vein of a full-grown dog; in *four seconds* the animal became tetanic, and in twenty-eight minutes it died. In these cases the absorption of the poison was inferred from the physiological effects produced, and not from the chemical demonstration of its presence in the blood and tissues. The chemical results vary according to the dose of strychnia administered and the time during which the person survives, as well as other circumstances.

Dr. McAdam states that he detected strychnia in the tissues of a cat which died in *fifty-six minutes* after a quarter of a grain had been given; and he found it in the urine voided by a dog only *nine minutes* after half a grain had been given to the animal. The dog was not at the time suffering from symptoms of strychnia-poisoning. This result shows that absorption takes place rapidly, and that the urine is a medium of elimination even before symptoms of poisoning are manifested. (*Guy's Hosp. Reports*, October, 1856, p. 393.) In a horse killed in *two hours* by thirty-two grains given in divided doses, Dr. McAdam found strychnia in the muscles, blood, and urine contained in the bladder. He did not detect it in the liver, lungs, spleen, kidneys, or heart. (*Pharmaceutical Journal*, August, 1856, p. 126.) It had not, therefore, been deposited in these organs within that time. Dr. Cowan, of Glasgow, poisoned three dogs, by giving to each of them one-quarter of a grain of strychnia. Dr. Anderson found traces of the poison in the liver of one dog, and Dr. Easton found it in the urine of another; but the time which they survived is not stated.

On the other hand, Dr. Harley, of University College, examined the blood taken from the heart and large vessels of a dog killed by the twelfth part of a grain of acetate of strychnia injected into the jugular vein. The animal was tetanic in *four seconds*, and died in twenty-eight minutes. The blood, on chemical analysis, yielded no strychnia. Mr. Horsley, of Cheltenham, examined the blood and tissues of a dog, which died in six hours after swallowing two grains of strychnia, but no strychnia could be detected in them. He sent me a portion of the blood of the dog, about two ounces, and, on analysis, I did not find in it any trace of strychnia. Dr. De Vrij, of Rotterdam, poisoned a dog with a solution of nitrate of strychnia introduced into a wound, and immediately after death he examined four ounces of the blood of the animal, but no trace of strychnia could be found in it. In another experiment in which a dog was poisoned in four days by half a grain of strychnia in divided doses, the chemical analysis led to a negative conclusion, not only in the blood and tissues but in all parts of the body. (*Pharmaceutical Journal*, March, 1857, p. 450.) Dr. Crawcour, of New Orleans, gave half a grain of strychnia to a rabbit; the animal died in half an hour. No trace of strychnia could be found in any part of the body. (*New Orleans Med. Gazette*, Sept. 1856, p. 387.) Dr. Penny, of Glasgow, examined the brain and spinal marrow of a dog, poisoned by strychnia, without detecting a trace of the poison. It has been asserted that strychnia is always eliminated by the urine; and experiments on animals, in reference to this point, have been cited. In a case of poisoning by strychnia, which occurred to the late Dr. Geoghegan, of Dublin, in 1856, thirty ounces of urine passed by the patient from the fifth to the thirty-first hour after symptoms had commenced, when carefully analyzed, did not yield any trace of strychnia. Dr. De Vrij examined the urine passed in twenty-four hours by a patient taking half a grain of nitrate of strychnia daily, medicinally, but he did not find in it any trace of the alkaloid. (*Pharm. Journ.*, March, 1857, p. 450.) A case of some interest occurred to Mr. Wilkins, of Newport, in the Isle of Wight, in February, 1857. A gentleman died, under the usual symptoms, in about *six hours* after taking three grains of strychnia for the purpose of self-destruction. The long period which he survived was most favorable for the diffusion and deposition of the poison. The blood and the heart were examined by the late Mr. Scanlan and myself; portions of the liver and lungs were examined by Dr. Christison and Dr. Douglas Maclagan, of Edinburgh; and one kidney was examined by Dr. Geoghegan, of Dublin. The result was, no trace of absorbed strychnia was detected in any one part. Observations made on the human subject do not, therefore, support the view that absorbed strychnia is either constantly eliminated by the urine or always deposited in the tissues so as to admit of separation by chemical processes after death.

It is quite clear, therefore, from the negative results obtained by gentlemen, many of whom could have had no intention to uphold a foregone conclusion, that strychnia is one of the alkaloids which in some cases is either speedily eliminated, or, if deposited in the tissues, is diffused in so small a quantity that the most refined chemical process at present known cannot separate it. To assert that the minutest quantity of this poison can always, and under all circumstances, be detected in the solids and fluids of the human body, because an almost infinitesimal quantity can be detected *out* of it, is not merely a simple absurdity, but an untruthful statement, calculated to mislead a jury and to deceive the public. Looking to what has been discovered respecting the absorption, deposition, and elimination of such poisons as arsenic and antimony (so easy of detection), it is only reasonable to suppose that strychnia is not an exception to the variations to which they are known to be subject—namely, that it may be found in one organ or secretion and not in another, and that at one time the body may yield evidence of its presence, while at another time there may be no such evidence forthcoming.

Since the publication of the former edition of this work, other cases have come before competent analysts with the like variable results. In one which occurred to Dr. Reese, of Philadelphia, involving a charge of murder, a woman lived five hours after taking a dose of strychnia. The body was not examined until six weeks after death, and the result was that no strychnia could be detected either in the contents of the stomach and intestines, or deposited in the tissues. In September, 1869, a lady died about three hours after she had taken a dose of strychnia in solution. The stomach and liver were examined by Mr. Horsley, of Cheltenham, but no strychnia could be detected in them.

In April, 1864, I was consulted by Dr. Edwards in the following case: A man æt. 43 swallowed, by mistake, five grains of strychnia rendered quite soluble by admixture with orange-juice. Tetanic symptoms soon came on in a violent form, and he died in a little more than *half an hour*. Dr. Edwards found about a grain of the alkaloid in the stomach: there was also a portion of it on the tongue. This was, no doubt, the unabsorbed poison. He also found it in the liver. I detected strychnia in eight ounces of the liver, but there was none in the kidney, nor in six ounces of blood. In a case which occurred to the late Prof. Casper, of Berlin, a man died in *three hours and a half* from a dose of five grains of strychnia. Three grains were procured from the stomach (unabsorbed), but none was found in the blood or deposited in the tissues. It is obvious in this case that two grains only could have been removed by absorption, leaving but a small quantity for deposition in the tissues.

The smallness of the quantity may sometimes explain the negative results. In an experiment in which a rabbit was killed in twenty minutes, by one-sixteenth of a grain of strychnia applied to the cellular membrane, no trace of the poison could be detected in the heart, liver, or blood. These negative results do not show that strychnia is not absorbed and deposited like other poisons, but simply that under certain conditions it cannot be detected in the organs of the body, in a case in which beyond doubt it has destroyed life.

As absorption and elimination cease at the time of death, the detection of an absorbed substance in the body will depend, *cæteris paribus*, on the length of time which a person survives after taking it. Dr.

Dupré found in some experiments on the alkaloid *quinia* that it was entirely eliminated from the body in two days.

In reference to the detection of the other alkaloids in an absorbed state, there is an absence of facts. That they enter the blood by absorption is, physiologically speaking, placed beyond doubt; but whether, when there, they are partially changed, or deposited unchanged in the organs, has not yet been satisfactorily established by experiment. I have elsewhere published some observations on this subject (*Guy's Hospital Reports*, Oct. 1856), and the researches of Dr. De Vrij, of Rotterdam, have more recently led him to the conclusion that that part of the alkaloid strychnia which acts mortally, is decomposed in the living body (*Pharm. Journal*, March, 1857, 451); and the same may be true of other alkaloids.

M. Bussy found, in giving to a dog an aqueous solution of extract of belladonna (*atropia*), that in *fifteen minutes* there was a perceptible dilatation of the pupils of the eyes—a clear proof that atropia had been absorbed and had so far saturated the blood as to paralyze the ciliary nerves. (*Ann. d'Hygiène*, 1847, vol. 2, p. 418.)

Dr. Burman, in referring to the alkaloid *conia*, quotes some experiments of Zalewski, which show that the poison, after being given to an animal, speedily appears in the urine, and is constantly present in that secretion during the progress of the toxic symptoms. The alkaloid is excreted entirely through the kidneys, having been detected in the urine of a dog two and a half days after the administration of the poison. (*On Conia*, 1872, p. 35.) MM. Voisin and Louisville have made a similar observation respecting *curarina*. They found that this poison was speedily eliminated in the urine of animals which had been poisoned by the hypodermic injection of curara. It was discovered, not only chemically, but physiologically, as they found that the urine of one animal injected into a wound, caused the death of another under the symptoms of curara poisoning. They also found that the urine of an animal poisoned with curara contained sugar. (*Ann. d'Hyg.*, 1866, vol. 2, p. 155.)

In a remarkable case of poisoning by *nicotina*, in Belgium, in 1847, M. Stas announced the discovery of this alkaloid in the tissues; but it is questionable whether, from the parts in which it was found, this was not some portion of the nicotina which had been imbibed by the organs, rather than that which had been absorbed and deposited in them. No cases are given, or facts mentioned, which will enable us to fix the time for absorption, deposition, and elimination; but M. Stas makes this general statement: "I have applied the principles just laid down (by his method of research) to morphia, codeia, strychnia, brucia, veratria, emetina, colchicina, aconitina, atropia, and hyoscyamia; and I have been able, without the slightest difficulty, to separate these different alkaloids when previously mixed with foreign matters." These results, however, cannot be taken as referring to the separation of the poisons above mentioned (deposited as a result of absorption) from the viscera of human beings or animals which had taken them during life; for on this subject there is no account of a single experiment. The analysis refers to the separation "of strychnia and brucia from nux vomica, veratria from the extract of veratrum, emetina from the extract of ipecacuanha, colchicum from the wine of colchicum, aconitina from an aqueous extract of monkshood, hyoscyamia from a very old extract of henbane, and finally atropia from an old tincture of belladonna. (Flandin, *Traité des Poisons*, vol. 3, pp. 134 and 255, 1853.) Facts of this description have only a pharmaceutical interest, for until the results have been verified by repeated analyses of the organs of persons poisoned by the different substances, and dying at long or short intervals, they are of very little value to a medical jurist. Some of the poisons which M. Stas mentions, will destroy life in a minute fractional proportion of a grain; and no process, however delicate, can make up for a very small quantity of poison distributed by the circulation through an enormous mass of animal matter.

CHAPTER VII.

ELIMINATION OF ORGANIC POISONS—SERPENT POISON—RABIES—INSECT POISONS—ELIMINATION BY THE BILE, SALIVA, AND MILK—THROUGH THE SECRETIONS OF SEROUS AND MUCOUS MEMBRANES—TRANSFERENCE OF POISONS FROM THE SKIN TO THE STOMACH AND INTESTINES.

Serpent Poison.—It has been elsewhere stated (p. 23) that this poison undergoes absorption, although, as it is emitted from the serpent in a wound, it may be truly said to operate by injection into the blood. Although a neurotic poison, a fact established by its action on the nervous system, it possesses, according to Dr. Fayrer, local irritant properties, for when applied to the mucous membrane of the eye it caused violent inflammation, with swelling of the eyelids. He states that persons bitten by the cobra generally complain of a severe or burning pain in the part, and this is followed by swelling and lividity of the surrounding skin, and in some instances by gangrene of the skin and subjacent cellular tissue, with other changes indicative of general blood-poisoning. (*Thanatophidia*, p. 36.) The poison itself is a glairy colorless viscid liquid, almost neutral in reaction. It has the property of destroying by contact the irritability of the voluntary muscles, and as it is injected from the serpent's tooth, it has a septic effect, the muscles having a tendency to undergo rapid decomposition.

This powerful organic poison is subject to elimination. Dr. Fayrer states that it is excreted by the kidneys and mammary glands, and probably also by the salivary glands and mucous membrane of the stomach. It has produced fatal effects on a child by its elimination through the milk (p. 55). Its passage into the urine by the kidneys was demonstrated by an experiment performed by Mr. Richards, of Balasore, who found that some *urine* from a dog, poisoned by the bite of a sea-snake (*Euhydrina Bengalensis*) killed a pigeon in twenty-two hours after being hypodermically injected. This gentleman also proved that the saliva was a medium of elimination. He found that one drachm of a greenish-colored saliva, which flowed from the mouth of a dog poisoned by cobra-venom, killed a pigeon in two hours. At the time the fluid flowed from its mouth the animal was paralyzed and motionless. (*Proc. R. S.*, Jan. 1874, p. 129.)

Rabies.—In reference to the poison of rabies, this appears to be transmitted entirely by the saliva of the dog; but whether it arises from spontaneous changes in the saliva or whether this secretion is a medium of elimination for an organic poison generated by disease in the animal, has not been determined.

The most remarkable feature of this poison is that its effects are so slowly produced. My colleague, Mr. J. C. Forster, who has given some attention to the subject of hydrophobia in man, finds that out of thirteen cases which he had collated, the shortest time that elapsed between the bite and the appearance of the disease, was four weeks, and the longest—in one case only—five to seven years! In the remaining eleven cases the disease showed itself at various periods within eleven months—the most protracted cases being those in which the persons had been bitten through the clothes. Comparing this with other animal poisons, he states that syphilis never exceeds a month from the time of contact to the appearance of the symptoms. The pyæmic poison ceases to be dangerous so soon as its source is removed, but in hydrophobia the poison may be dormant in the system for at least a year before showing its effects! From his observations the common belief that pain and irritation are felt in the wound before the attack is erroneous. Local irritation was observed in only one out of thirteen cases; but there was pain in the course of the nerves leading from the injured part. This was one of the most marked symptoms of the commencement of the attack. The other striking symptom was not the dread of liquids, but the inability to perform the act of swallowing when the liquid was taken into the mouth. (*Guy's Hosp. Rep.*, 1866, pp. 18, 21.)

From the length of time required for the production of symptoms, it might be inferred that absorption was entirely suspended in reference to the poison of rabies, and therefore that the early removal of the bitten part would insure safety; but the disease has shown itself even where this practice has been adopted. Assuming that the poison is absorbed and diffused by the blood, it appears to require a long time for incubation in the blood before producing the symptoms of the disease.

The animal poisons of the wasp and bee are strongly acid, owing, it is supposed, to the presence of formic acid. Although the quantity injected is infinitesimally small, this poison causes the most severe local pain and swelling, and in some instances, these local effects are followed by syncope and great constitutional disturbance. In the *Lancet*, for 1872 (vol. 2, p. 135), is reported the case of a lady, æt. 55, who died apparently from shock after she had been stung by a bee behind the ear. It seems that she was a woman of a highly nervous temperament, and that she became unconscious soon after the sting. A similar case occurred in August, 1874. A woman, æt. 50, was stung by a hornet. She fainted, and died from shock soon afterwards. Inquests were held in both cases.

There is nothing to show that this poison undergoes absorption. The quantity injected into the minute wound produced by the sting of the insect is too small to admit of being traced beyond the wounded spot. As with the serpent-poison, the effects are produced by injection into the blood.

Elimination has been hitherto considered chiefly as it takes place by the *urine*. Bile, saliva, and milk, as well as the mucous and serous secretions of the body, are also media by which poisons are ejected. Metallic poisons which are deposited in the liver, pass off through the bile. Mercury is especially eliminated in the saliva, and arsenic and antimony in mucous and serous liquids.

The milk has been but little examined for the presence of poisons. In one instance, in which a cow suffered from the effects of lead-poisoning, the animal having licked up a quantity of white paint, I found traces of lead in the milk a few hours after the poison had been swallowed. (*Guy's Hosp. Reports*, 1841, No. 12.) It is a well-known fact, in reference to a woman while suckling, that medicinal and noxious substances are conveyed rapidly by the milk into the body of the child, and may seriously affect it (p. 37). A case is quoted by Sir R. Christison, which will serve as an illustration. It occurred to M. Minaret, a French physician. A young woman, who was taking medicinal doses of tartar emetic for pleurisy, suckled her infant, and it was observed that the child was attacked with a fit of vomiting immediately after every attempt to suck the breast. (*On Poisons*, p. 483.)

Even the serpent-poison, according to Dr. Fayrer, is eliminated by the mammary glands, and passes off with the milk. In proof of this statement, he quotes a case reported by Mr. Shercore, of Calcutta. "An infant was suckled by its mother after she had been bitten by a venomous snake of unknown species. The child died in two hours after it had partaken of the milk, evidently from the effects of the poison." It is remarkable that the child took the breast before any marked symptoms of poisoning had occurred in the mother. (*Thanatophidia*, p. 43.) This case furnishes a proof that serpent-poison may be absorbed by the gastric mucous membrane of an infant in sufficient quantity to cause death.

M. Jacquemin examined the milk of a cow which had been severely wounded while at pasture. The wound had been dressed with carbolic acid. He states that he detected carbolic acid in the milk drawn from the cow. (*Pharm. Journ.*, 1874, April 25, p. 852.)

Mr. Steele states that two ewes were bitten by a rabid dog. Rabies appeared in them about six weeks afterwards and they were killed. One had two lambs, the other one. At first, these lambs were permitted to suckle. They were subsequently attacked with rabies, and were then killed. It appears highly probable that they received the poison through the milk, because they were removed from the ewes a month before these became affected; there was no mark of their having been bitten, nor is it proved that a sheep can communicate the poison by a bite, either before or after it has been attacked with rabies. (*Med. Gaz.*, vol. 25, p. 160.)

These facts are sufficient to show that mineral and organic poisons escape from the body through the milk.

Serous Elimination.—In a case of poisoning with arsenuretted hydrogen, Dr. O'Reilly examined a quantity of reddish-colored liquid which had been effused in the chest, and he found in it arsenic. This proves that arsenic is eliminated in the liquid effused from serous membranes.

Dr. Chatin has applied these results practically as an additional aid to diagnosis in a case of poisoning with arsenic. He applied a blister to the chest of a woman suffering from the effects of arsenic. He collected ten drachms of serum from the blister, and he obtained from the arsenic contained in it sixteen well-marked metallic deposits by the use of Marsh's process. (*Journal de Chimie*, 1847, p. 329.)

Mucous Elimination.—This has been especially noticed with respect to arsenic and antimony. In a preceding page, the diffusion of arsenic in the body by the mucous secretions has been already noticed. To some, who have not considered this question in all its bearings, it may appear a startling proposition to make—that arsenic may be found after death in the stomach of a person who has not taken any of the poison by the mouth. This fact, unless explained, might wrongly involve an innocent person in a charge of criminal poisoning, and it might lead to an erroneous inference respecting the time at which poison had been taken or administered. Orfila found arsenic in the stomach and intestines of a dog which had been killed in four hours by the application of three grains of the poison to the cellular tissue. The late Dr. Brinton injected ten grains of tartar emetic dissolved in water into the femoral vein of a dog. At the end of fifteen minutes the animal was killed, and the contents of the stomach, then in the act of digestion, were examined. They were found to contain antimony in rather large proportion. This proved that the poison was not only transferred from the thigh to the stomach, but that it was rather rapidly transferred to, and accumulated in, that organ.

Dr. Pavy and I performed experiments on dogs, in order to test the accuracy of this theory of transference. Solutions of tartar emetic, varying from two to six grains, were injected into the jugular veins of three dogs. The animals died in from eight hours to thirty. Antimony was found in each case in the contents of the stomach and intestines, but always in small proportion. Other experiments performed with a solution of arsenic were attended with similar results. One animal died in ten hours after the injection of one grain, and another in eighteen hours after the injection of two grains of arsenic in solution. In a third experiment, the mixed poisons were injected, and the animal died in twelve hours. Arsenic and antimony were found in the fluids of the stomach and intestines in each experiment. (*Guy's Hosp. Reports*, 1860, p. 397.)

Dr. Fraser obtained results of a similar kind in his experiments with the extract of Calabar bean. He injected five grains of the extract dissolved into the jugular vein of a dog. The animal died in eleven minutes. An extract was made of the contents of the stomach, and a small portion applied to the conjunctiva of a rabbit. The well-known physiological effect of this substance was soon manifested. The pupil strongly contracted and remained so for an hour. (*Physiological Action of the Calabar Bean*, 1867, p. 4.)

These facts connected with mucous elimination convey a warning to medical witnesses who rely strongly upon the detection of *traces* of poison in the stomach and intestines as a proof that the poison has necessarily been administered or taken by the mouth. The detection of poison in these parts simply shows that it must have entered the body by some channel, either by the mouth or by the skin. In these cases the amount of poison found is always very small, and always in solution in the fluids. If it be found in lumps, or powder, or largely dissolved in the liquids of the stomach, these conditions would be inconsistent with mucous elimination.

In February, 1864, the following case was remitted to me for examination by Secretary Sir George Grey. A girl æt. nine, the daughter of a man named Bootman, died after a short illness without medical attendance. The cause of death was obscure: the symptoms resembled those of arsenic, but there was no evidence of administration in food, and the girl died from exhaustion, only after nine days. It turned out that a day or two before the fatal illness set in, the stepmother had rubbed a portion of white precipitate ointment into the scalp of the child to destroy vermin. Some white arsenic had been unknowingly mixed with this ointment. This had caused the child's death by absorption. Arsenic was found in the scalp and in the mucous fluids of the stomach and intestines, as well as the liver; but only in very small quantity, and in a dissolved form.

The woman was suspected of having destroyed the child intentionally. She was a stepmother, and was reported to have illtreated the child on various occasions. The question before the coroner's jury was: As poison was found in the stomach, did she give to the deceased any arsenic in her food? They were inclined to adopt this view, but the presence of arsenic in the stomach and intestines was ascribed simply and entirely to the elimination of the poison by the mucous secretions; and a verdict was returned accordingly. (*Guy's Hosp. Reports*, 1864, p. 220.) The arsenic found was in traces, perfectly dissolved in the fluids. The symptoms were slow in appearing, and at no time urgent, and the case only proved fatal after nine days. These facts were consistent with the introduction of the poison by the skin. At an earlier date the detection of the poison in the stomach in such a case might have led to a conviction for murder.

It is strange that mucous elimination by the stomach and the fallacies to which it may give rise should have been so long overlooked by medical jurists, because it had been generally understood that the saliva was a medium for elimination, and this, as we know, is a mucous secretion.

From these facts it must not be supposed that so long as arsenic or antimony remains in any part of the body, it will be found in the stomach. After death it may be detected in the liver and kidneys, and not in the contents of the stomach and intestines. In the case of McMullen (Liverpool Summer Ass., 1856), Mr. Watson found no antimony in the stomach or contents, while it was most abundant in the liver, spleen, and the kidneys. Like arsenic it is not found in equal proportion in all the solid organs or in all the fluid secretions. One may contain it and another not.

CHAPTER VIII.

REMOTE OR SYSTEMIC ACTION OF POISONS—ORGANS SPECIALLY AFFECTED—CAUSE OF DEATH—PROPORTION OF POISON CONTAINED IN THE BLOOD IN FATAL CASES—PHYSICAL AND CHEMICAL CHANGES PRODUCED IN THE BLOOD BY POISONS—CHANGES PRODUCED IN CERTAIN POISONS—SPECTRAL ANALYSIS OF POISONED BLOOD—ANTAGONISTIC POISONS—TREATMENT OF CASES OF POISONING—ALLEGED ANTIDOTES—GENERAL CONCLUSIONS.

Remote or Systemic Action of Poisons.—By this we are to understand that power which most poisons possess of affecting some organ or organs remote from the part to which they are applied. The same substance often possesses both a local and remote action, but some poisons affect one organ remotely, and others another. Cantharides, a poison which has a violent local action as an irritant, to whatever part of the body it may be applied, affects remotely the urinary and generative organs. Mercury affects the salivary glands. Morphia, whether applied to a wound or to the mucous membrane of the stomach, affects the brain. Digitalis taken internally affects the heart; strychnia, the upper part of the spinal marrow; prussic acid, the brain and spinal marrow. Belladonna produces a dilatation of the pupils by paralyzing the ciliary nerves, and it produces this effect whether applied locally to the eye or taken into the stomach. MM. Kölliker and Pelikan state that the *Tanghinia*, or poison of Madagascar, has a paralyzing action on the heart and muscular system especially. It paralyzes the nerves; but they regard it as essentially a muscular poison. (*Proc. of Royal Society*, No. 30, vol. 9, p. 174.) The curara poison, when it acts rapidly, destroys life without producing convulsions, and exerts a special paralyzing influence on the nervous system. It acts in a mode precisely the reverse of strychnia. It destroys the nervous system from the circumference to the centre, while strychnia, in producing violent convulsions, destroys it from the centre to the circumference. Strychnia acts upon the nerves of motion and sensation. It frequently exalts sensibility to a very high degree. Curarina, the alkaloid of curara, operates only by paralyzing the nerves of motion, the paralysis of those of sensation being simply a consequence of the asphyxia resulting from the cessation of respiration. But while curara paralyzes the nerves of motion, it does not destroy the contractility of the involuntary muscles; the heart continues to beat in animals poisoned by it. (*Bernard*, *op. cit.*, 316, 341, 346.) This remarkable poison allows of an entire separation of the two functions of the nervous system,—motion and sensation. In the action of chloroform a converse effect has been noticed: there has been a complete paralysis of sensation, while the nerves of motion have retained their power.

Strychnia appears to exert no poisonous action, or but a slight effect, on animals destitute of spinal marrow. Bernard has made this observation on leeches, and I have found that the larvæ of insects may be immersed in a strong solution of strychnia, or even covered with finely powdered acetate of strychnia, without any indication of the effects produced on vertebrated animals by this poison. Aconite has both a local and a remote action. The root when chewed causes a peculiar tingling and numbness of the lips. Its remote action as a result of absorption, is manifested chiefly on the nerves of sensation. The late Dr. Pereira found that an alcoholic extract of the root produced complete loss of sensibility in a dog, although the animal was able to walk. (*Mat. Med.*, vol. 2, part 2, p. 686.) Dr. Fayrer noticed the reverse effects with the cobra-poison. The sensory nerves were but little affected by it; they retained their power after the motor nerves were paralyzed. This poison also caused paralysis of the reflex function of the cord. In some cases, this action is more obscure; and the same poison will affect remote organs differently, according to the form and quantity in which it may have been taken and perhaps according to peculiarity of constitution in the person. Conia (the poison of hemlock) paralyzes the motor nerves as well as the spinal cord; but Dr. Fraser noticed that this remote action differed according to the dose. When small, the motor nerves were paralyzed before the reflex function of the cord, but when large, the cord was paralyzed before the nerves. So with regard to the cobra-poison, Dr. Fayrer found that its action on the heart depended on the dose. Under small doses, the heart continued to pulsate vigorously long after all motion had ceased in the voluntary muscles, and the strongest irritation to the spinal cord and motor nerves produced no effect. When, however, a large quantity of cobra-poison was introduced at once into the circulation or absorbed with great rapidity, the action of the heart was at once arrested. It was not paralysis but tetanic contraction of the heart which was produced, the poison, in fact, seeming to act as an excessive stimulus. (*Op. cit.*, p. 122.)

The Calabar bean is a cardiac poison; in small doses it diminishes the pulsations of the heart, under a large dose the animal dies at once from paralysis of the heart. It has no action on the brain. Sir R. Christison maintained his consciousness and mental vigor while suffering from the effects of this poison on the heart. The cobra-poison has no direct action on the brain. Dr. Fayrer observed that intelligence was retained until the last.

The mineral acids rarely affect the brain remotely; the mental faculties, in cases of poisoning by them, commonly continue clear until the last moment of life. Arsenic sometimes affects the heart—this is indicated by syncope; at other times the brain and spinal marrow—this is known by the coma, stupor, numbness, tingling, and paralysis of the extremities that occasionally supervene in poisoning by this substance. In other cases its effects have been chiefly manifested on the spinal marrow, indicated by violent tetanic convulsions. Oxalic acid was found by Christison and Coindet to affect remotely either the heart, the spinal marrow, or the brain, according to the strength of the solution in which it was administered to animals.

In all cases of *acute* poisoning, *i.e.* cases in which the symptoms run through their course rapidly—whether the substance has a local action or not—death is commonly referable to the influence exerted by the poison on a remote organ important to life. Most poisons destroy life by affecting the heart, brain, or spinal marrow. The impression produced on either of these important organs is, however, not always so intense as to kill; for individuals have been known to recover from morphia, strychnia, or prussic acid, even after alarming symptoms, as a result of this remote influence, had manifested themselves. In some instances, however, the impression produced is such as to annihilate speedily the vital functions. Thus large doses of prussic acid, conia, or strychnia may destroy life in a few seconds or minutes, without producing any perceptible local changes on the body.

Cause of Death.—When a poison like concentrated sulphuric acid proves rapidly fatal, without entering the blood by absorption, death is ascribed to the shock impressed on the general nervous system from the effects of the poison on the living tissues. The nature of the fatal impression thus produced, can no more be determined than the nature of thought or sensation. There is, however, no greater difficulty in conceiving that such an impression may be excited by a poison, than that a slight mechanical injury in a remote part of the body may cause an attack of tetanus. (*Addison and Morgan on Poisonous Agents*, p. 64.) The fact that the greater number of poisons enter the blood and act fatally through the medium of this fluid, does not bring us any nearer to an explanation of the direct cause of death. One hypothesis assumes that the organ remotely affected, is poisoned by the blood which contains the substance dissolved. The doctrine was supported by Liebig in a modified form. He considered that an alkaloidal poison—morphia, for example—might be chemically converted into a substance like brain by the subtraction of some elements and the addition of others, the quality of the cerebral matter becoming thereby changed, and rendered unfit to support vital energy.

The most remarkable fact connected with the diffusion of poison in the blood is the small amount which is required for the destruction of life. This may be tested by the smallest fatal doses of some well-known substances. In one well-observed case, two grains of arsenic, given over a period of five days, destroyed the life of an adult. Supposing the whole of this quantity had entered into and remained in the blood, it would have formed only the 98,000th part by weight of that liquid, but as elimination and deposition go on simultaneously, the proportion actually in the blood at any given time must have been much less than this; and yet there can be no doubt that the poison destroyed life by its action on the blood! Half a grain of strychnia has destroyed the life of an adult in twenty minutes. Admitting that the whole was absorbed and equally diffused in the blood, it would have amounted only to the 392,000th part by weight. A child has thus been destroyed by the sixteenth part of a grain. Assuming that all was absorbed and retained in the four hours during which the child survived, the proportion held by the blood would have been only the 1,344,000th part. The lethal principle in serpent-poison would probably form a much smaller proportion than any of those here given. From Dr. Fayrer's experiments on cobra-poison, there is reason to believe that the greater part is retained in the blood. (*Proc. R. S.*, Jan., 1874, p. 132.) But the quantity there present at any one time is infinitesimally small. Still, it is sufficient to render the blood poisonous to other animals.

Changes Produced in the Blood.—There are few physiologists who doubt that all absorbed poisons act through the blood, and that they alter its physical or its chemical properties; sometimes manifested by an alteration in its consistency, or by a change of color—a portion of the poisonous substance itself simultaneously undergoing a change. In some instances the blood is rendered directly poisonous to other animals, producing symptoms and death like the original poison. This is remarkably illustrated by the effects of the cobra-poison, the presence of which does not admit of any chemical demonstration. Dr. Fayrer found that a few drops of the blood of a dog killed by the bite of a cobra caused death in seventy-five minutes when injected into the thigh of a fowl. (*Thanatophidia*, pp. 80–83.) He considers from this that the removal of the poisoned blood and the substitution of healthy blood for it by transfusion, would form a rational mode of treatment.

Among early observations on the chemical changes produced by poisons in the blood are those of Sir R. Christison on the effects produced by oxalic acid. He could not discover any oxalic acid in the vena cava of a dog which had died in thirty-six seconds from the injection of eight and a half grains of that poison into the femoral vein. (*On Poisons*, p. 18.) Bernard also announced the conversion of cyanide of mercury into hydrocyanic acid while traversing the capillary system of the lungs. (*Op. cit.*, p. 66.) Organic poisons may undergo similar changes, although this is a matter of inference rather than of proof. With some, however, it admits of demonstration.

The vapors of chloroform and ether, nitrous oxide, sulphuretted hydrogen, carbonic acid, and other gases darken the blood. The vegetable alkaloids, strychnia and morphia, darken it and render it fluid. This darkening of the blood has been in some cases ascribed to the effects of induced asphyxia, as on re-exposure to the air the blood has again become florid red. Oxalic acid gives to it a dark brown color, anilin a crimson purple, and prussic acid in some cases a dark purple.

Dr. Fraser observed that the blood obtained from animals which had been poisoned by the Calabar bean (physostigmia) was generally dark in color; but when drawn from the left side of the heart after a very large dose of the poison, it had the scarlet hue of arterial blood. It frequently remained semifluid for some time and then clotted loosely. In dogs and rabbits the red blood-corpuscles were changed in form, and presented various irregularities of outline, among which a well-marked stellar crenation preponderated. (*On Calabar Bean*, p. 55.) Fontana long ago stated that the serpent-poison darkened the blood and prevented coagulation. It appears, from more recent observations, that the cobra-poison kills without destroying the coagulability of the blood, while the poison of the daboia—another Indian serpent—causes in the blood perfect and permanent fluidity. The blood was no doubt altered, but no corpuscular changes could be detected in it.

Arsenic, antimony, corrosive sublimate, and the greater number of mineral and metallic poisons, produce no change in color or physical properties. Although we are unable to prove by experiment what chemical changes the blood itself undergoes, it is easy to show, with respect to some of these substances, that they are themselves partially converted into other bodies while circulating, and this can only take place at the expense of the constituents of the blood.

There is reason to believe that hydrate of chloral is partially converted into chloroform in the blood (*Wiggers Jahresber*, 1871, p. 566), and in reference to chloroform itself, it has been found in cases in which the vapor has proved fatal, that a portion of it was converted into formic acid. The blood had also lost the property of coagulation, and of becoming florid red by exposure to air. (*Chemist*, 1856, p. 544.) Nitrobenzole is converted into anilin—the essential oil of bitter almonds into hippuric and benzoic acids. Oxalic acid is probably partly converted into carbonic acid and carbonic oxide, but a portion may be eliminated in the urine as oxalate of lime. Alcohol, ether, prussic acid, conia, and nicotina, from the changes produced in color and consistency of the blood, are most probably converted into new compounds not yet isolated. In all these cases the conversion is only partial, for the separation of a portion of the poison from the blood proves that some is unchanged. There is an exception to this remark in oxalic acid, which has not been detected as such in the blood in cases of poisoning by this substance, even where it has been injected into the vein of a living animal. (Christison, *On Poisons*, p. 18.)

Some of the chemical and physical changes abovementioned indicate that the absorbed substances have either removed from the blood oxygen (ozone), or have in some way neutralized it. It is a remarkable fact that some of the most powerful poisons, such as prussic acid, conia, and nicotina, contain no oxygen, and under certain conditions they manifest a strong tendency to combine with it.

The presence of oxygen (ozone) in the blood of the arterial capillary system is indispensable to innervation, the metamorphosis of tissue, and all those changes which create the marked distinction between life and death. It is a question yet to be solved whether the lethal action of these poisons may not be in part due to the suspension of these oxidation changes.

According to Rossbach, the alkaloids strychnia, veratria, digitalia, atropia and others exert a poisonous action by forming compounds with albumen and arresting those oxidation changes in the blood to which albuminous substances are liable during life. Albuminates of the alkaloids are produced which are less soluble than albumen, and which have not the same power of absorbing or fixing oxygen. The hæmoglobin or albuminous coloring liquid contained in the blood-cells produces and transmits ozone, in spite of these alkaloids; but they have the property of more completely fixing ozone in the hæmoglobin, and thus they prevent it from being so readily transmitted to other bodies, a process necessary to the maintenance of the vital forces. (Bouchardat, *Ann. de Thérapeut.*, 1874, p. 327.)

There is no evidence that mineral poisons, such as arsenic, antimony, or mercury, undergo any chemical changes in the blood.

Spectral analysis has been applied to the examination of poisoned blood; but even this delicate method of research has failed to throw any satisfactory light on the changes produced by poisons in this liquid. M. Preyer, of Jena, has performed a few experiments on the subject, but the results chiefly show the well-known changes in the absorption-bands, produced by the oxidation and deoxidation of the red coloring matter. He has delineated some of these spectra in contrast with those of normal blood. (*Die Blutkrystalle: Untersuchungen von W. Preyer.* Jena, 1871.)

The Prussic acid spectrum of blood represented in No. 12, Taf. 2, p. 231, presents two well-marked absorption-bands, which in size and position scarcely differ from those of normal blood. It was produced from a combination of a solution of cyanide of potassium with deoxidized coloring matter—(Cyanwasserstoff-sauerstoff hæmoglobin). There is a larger absorption of the violet and red rays than in the normal blood-spectrum. In the oxidized mixture of blood and prussic acid, or of alkaline cyanide, the two bands are merged into one broad band, with a more complete absorption of the violet and the blue, as well as a diminished absorption of the red rays.

The spectrum of oxidized blood with a large proportion of *oxalic acid*, forming a solution of a brown-red color, exhibits one band in the orange to the left of the sodium line, and a complete absorption of the violet, indigo, blue, green, and the greater part of the red rays. (No. 2, Taf. 2.) By dilution and admixture with various substances, this spectrum undergoes some changes; but the oxalic acid blood-spectrum was found to be similar to that of many other acids when mixed with normal blood.

Carbonic oxide, which gives to the blood a light red color, produces a spectrum with two absorption-bands similar to those of normal blood. The red and violet rays are more completely absorbed than in the spectra of normal blood, but with a strong solution of the red coloring matter the results are similar.

The alkaline sulphides (sodium and ammonium) as well as hydrosulphuric and carbonic acids simply act as reducing agents and give the spectra of deoxidized blood.

In mixing with blood, solutions of arsenic and other poisons which did not act chemically upon that liquid, I have not observed under the spectroscope any marked difference in the spectra from those of normal blood. Indeed, when it is considered how small a proportion of poison contained in blood is sufficient to destroy life, it is not surprising that negative results should be obtained (*ante*, pp. 60, 61).

Dr. Fraser states that he has examined spectroscopically the blood of animals which have died from the effects of Calabar bean, and that he has not observed any modification in the characters or position of the normal bands of absorption. (*Op. cit.*, p. 55.)

Antagonistic Poisons.—In their remote action on the body through the blood, some of these powerful agents appear to be antagonistic, and it has been supposed that they might be beneficially employed as counter-agents to each other. Thus it has been assumed that as strychnia is a powerful excitant of the nervous system, a poison like curarina, which depresses or annihilates nervous power, would be an efficient antidote. Bernard remarks that the convulsions caused by strychnia might be thus suppressed, but death would take place from such a mixture as certainly and as speedily as if curarina had not been given. Animals, according to him, have died even more rapidly in these experiments than when strychnia or curarina was given separately; although convulsions were suppressed when the two poisons were given at once. (*Op. cit.*, pp. 53, 377.) I agree with this physiologist in thinking that there is an absence of proof that these energetic substances can neutralize each other in the blood with safety to the patient, and that such a mode of treatment must necessarily be attended with danger. Nicotina, aconitina, and physostigmia (Calabar bean) have been employed, as well as curarina, as counter-agents to strychnia. Nicotina destroys muscular contractility, curarina paralyzes the motor nerves, and physostigmia arrests the functions of the spinal cord. (Fraser, *On the Calabar Bean*, p. 27.) Dr. Fraser has pointed out that there is an inherent difficulty in this mode of treatment. There are certain limits within which this physiological antagonism in the blood may be exerted, depending on the dose of absorbed poison to be counteracted, which is in all cases an unknown quantity. Beyond these, death may be produced by combined doses of the two substances, either by some non-antagonized action belonging to one or the other of them, or by a combination of similar actions belonging to both. (*On Physostigmia and Atropia*, 1871, p. 4.) Atropia has been proposed as an antidote to morphia, and physostigmia to atropia, apparently because they produce opposite effects on the pupils; but this action on the iris is not sufficient to justify the use of such powerful agents as antidotes to each other. If the poison has been taken in a large dose, the antagonistic treatment will be useless; and, if in a small dose, it may, as Dr. Fraser remarks, hasten and render more certain a previously doubtful fatal result. (*On the Calabar Bean*, p. 28.)

Attempts have been made, on similar principles, to antagonize animal poisons, such as that of rabies and the serpent-poison. Injections of curara, of atropia, and morphia, have been recommended for the treatment of hydrophobia, but there is no evidence to show that these have proved beneficial. My colleague, Mr. J. C. Forster, has used atropia by injection to counteract the effects of the poison of hydrophobia in man, and for eleven hours, during which it was employed, a marked quiescent condition appeared; but, after the injection of the thirty-sixth of a grain, the man rapidly sank, whether from the effects of the atropia or the hydrophobia, or both combined, it would be difficult to say. (*Guy's Hosp. Rep.*, 1866, p. 24.)

The only rational mode of treatment is to prevent absorption, and promote elimination. Future experiments may show that the poison already in the blood may be neutralized with safety to the patient by the injection of other liquids as counter-poisons; but, at present, satisfactory evidence on this point is wanting. Ammonia injected into the veins or the cellular tissue was supposed to be an antidote to the serpent-poison; and it is stated that in Australia the use of this alkali had been proved to be a most successful mode of treatment. Experiments recently made for the Indian Government by Dr. Fayrer and others have clearly shown that ammonia is not an antidote in any correct sense of the word. In their carefully conducted experiments no benefit resulted from its use. (*Thanatophidia and Proc. R. S.*, Jan. 1874, p. 132.) It was then suggested that the Australian serpent-poison might be different from the Indian. To meet this objection, a tiger-snake was sent from Australia to India, and there experimented on by the committee. The result proved that the intra-venous injection of ammonia for the treatment of bitten animals, was utterly useless. The fallacy thus demonstrated in reference to the alleged antidotal properties of ammonia should teach caution in relying upon hypodermic or intra-venous injection as a method of treatment in other cases. It is obvious, from what is now known regarding the absorption of poisons, that any antidote, in order to be efficient, must be absorbed as rapidly as the poison itself, must follow it into the blood, and there neutralize its toxic properties, without in any way affecting the healthy properties of the blood!

Admitting that every poison could be chemically detected in the blood, it would yet remain to be explained *how* it operated when there to destroy life. At present there is no satisfactory theory to account for the fatal effect. All we know from observation is, that the poison when circulating through the bloodvessels destroys life; and all that we can say at present in reference to the cause of death is, that the blood is so changed by the poison as to render it unfitted to supply and maintain that amount of nerve-force which is absolutely necessary to the continuance of life. It may be expected that, in the progress of microscopical and chemical science, the precise effect produced by poisons on the blood will hereafter become a subject of demonstration; but, at present, the *modus operandi* is a perfect mystery. We trace the poison to the circulation, and we observe that death is the result; but neither the chemist nor the microscopist can throw any light upon the changes produced by the poison in the blood or in the organs necessary to life. The conclusions to which the foregoing observations lead are:

1. That all substances acting as poisons are carried into the blood, and no substance acts as a poison until it has been absorbed and circulated through the arterial capillary system.

2. The sooner the poison reaches the blood, either from its solubility or from the nature of the surface to which it is applied, the more rapidly does it produce its effects on the body.

3. The fatal effects of poisons depend not on the absolute quantity of the substance taken, but on the quantity absorbed within a given time.

4. Those substances which act by absorption are absorbed within a few seconds when placed under circumstances favorable to the process.

5. That elimination by the secretions and deposition in the organs commence so soon as a poison has entered the blood, and these processes continue until death, or in the event of recovery until all the poison has been thrown out of the body.

6. That the fatal effects depend on absorption taking place more rapidly than elimination.

7. That a large number of poisons susceptible of detection either by their chemical or their physiological properties, have been distinctly traced to the blood.

8. That with some not so traced, *e. g.*, the poison of venomous serpents, the blood and the fluids excreted from it, namely, the urine, milk, and saliva, act as poisons on other animals.

9. That the fatal proportion of poison present in the blood at any one time is infinitesimally small.

10. That unless speedily eliminated from the blood or deposited in the organs, poisons produce on this fluid such physical and chemical changes as to render it unfitted to maintain the functions of those organs which are necessary for the support of life.

11. That the poison found in the body after death is the surplus or residue of that which has been absorbed and eliminated, and has actually destroyed life.

12. That in some cases a person may die from the effects of a poison, although no trace of it may remain in the body at the time of death. Death takes place, not from the actual contact of the unabsorbed poison with the organs, but from the changes produced in the blood by that portion which has been absorbed.

CHAPTER IX.

INFLUENCE OF HABIT—TOLERANCE OF POISONS—OPIUM—ALCOHOL—TOBACCO—STRYCHNIA—ACTION OF ARSENIC—TOLERANCE OF ARSENIC AND ANTIMONY—ARSENIC-EATING—COSMETIC USES OF ARSENIC—IDIOSYNCRASY—INTOLERANCE OF POISONS.

Influence of Habit—Tolerance of Poisons.—It is a well-known fact that habit diminishes the effects of certain poisons. Thus it is that opium, when frequently taken by a person, loses its narcotic power after a time, and requires to be administered in a much larger dose. Indeed, confirmed opium-eaters have been enabled to take at once a quantity of this drug which would have infallibly killed them, had they commenced with it in the first instance. Even infants and children, who are well-known to be especially susceptible of the effects of opium, and are liable to be poisoned by very small doses, may, by the influence of habit, be brought to take the drug in very large quantities. This is illustrated by a statement made by the late Mr. Grainger, in the *Report of the Children's Employment Commission.* It appears that the system of drugging children with opium in the Factory districts commenced as soon after birth as possible; and the dose was gradually increased until the child took from fifteen to twenty drops of laudanum at once! This had the effect of throwing it into a lethargic stupor. Healthy children of the same age would be killed by a dose of five drops. This influence of habit is chiefly confined to poisons derived from the organic kingdom. It has been observed that the same influence is manifested in the use of tobacco, alcohol, ether, chloroform, morphia, strychnia, and other alkaloids. It is remarkable that poisons do not act upon all persons in a similar manner. The same dose may produce different effects in different persons, and so it may happen that the same poison will not operate in a similar manner or degree on man and animals. Some important medico-legal questions occasionally present themselves in reference to this subject. The tolerance of poisons may sometimes be traced to habit, to disease, or to peculiarity of constitution. The last condition will be considered under the head of idiosyncrasy.

The following cases will serve to illustrate the effect of habit in reference to the powerful poison, strychnia, whether taken by the mouth or administered by hypodermic injection:

1. This was the case of a woman, æt. 29, laboring under paralysis. She took, in pills, one-sixteenth of a grain of strychnia daily, and this was increased at intervals of four days, to one-eighth, one-half, and one grain daily. The dose was gradually raised to three grains daily, and this is stated to have been continued for six days! Tetanic convulsions of the limbs and other symptoms then appeared; the dose was gradually reduced. In two months this patient is said to have taken seventy grains of strychnia. (*Med. Gaz.*, vol. 36, p. 261; *Gazette Médicale,* Mai, 1845.) The bearing of this large quantity with comparative impunity, may have depended not only on habit, but on the diseased state of the system (tolerance) and on the rapid elimination of the poison.

2. Dr. Chisholm, U.S., using sulphate of strychnia by hypodermic injection into the cellular tissue, commenced with a dose of one-fortieth of a grain, increasing the daily dose gradually until it reached one-sixth or even one-fifth of a grain. In one case, the fourth of a grain was injected at once and continued daily without causing any muscular contractions or other unpleasant symptoms. The maximum dose was usually attained in from fifteen to twenty days. He safely used the injection of one-sixth of a grain daily for three months, making fifteen grains of sulphate or thirty fatal doses. This large quantity passed through the body without injury. (*American Journ. Med. Sci.,* Oct., 1872, p. 387.)

The only form in which I have known the question of habit raised in medical jurisprudence is this: Whether, while the more prominent effects of a poison are thereby diminished, the insidious or latent effects on the constitution are at the same time counteracted. The answer is of some importance in relation to the subject of life insurance, for the concealment of the practice of opium-eating by an insured person has already given rise to an action, in which medical evidence on the subject was rendered necessary. As a general principle, we must admit that habit cannot altogether counteract the insidious effects of organic poisons, and that the practice of taking them is liable to give rise to disease or impair the constitution. The habitual use of alcohol may enable a person to take this liquid daily in unusually large quantities, but it does not the less produce disease. The same remark applies equally to the daily use of opium and tobacco. If we believe that these narcotics are absorbed into the blood, and that, until eliminated, they arrest the oxidating processes of this liquid, we must admit that, however such effects may be reduced by habit, so long as they continue in any degree they must undermine health. If it be asserted that the effect of habit is to destroy this action on the blood, and to render the absorbed principles inert, the assertion is without proof or even probability; and in the case of alcohol it is clearly contrary to experience.

It has been hitherto considered by toxicologists that, except within very narrow limits, habit appears to exercise no influence on the action of *mineral* poisons. There is no proof that a human being has ever accustomed himself by habit, to take such substances as arsenic or corrosive sublimate in doses that would prove fatal to the generality of adults. It is well known in the medicinal use of arsenic that a slight increase in the dose has often been attended with such alarming symptoms as to render a discontinuance of the mineral absolutely necessary to the safety of the person. (See *Arsenic, post.*)

It is stated that in certain parts of Styria and Hungary, there are human beings who have so accustomed themselves to the use of arsenic, as to be able to take this substance not only without the usual symptoms of poisoning, but with actual benefit to health. This subject would hardly require serious notice in this place, but that it has already formed part of the medical evidence in some criminal trials for poisoning.

There is no reason to believe that arsenic-eating is practiced in this country; still an attempt may be occasionally made to turn this Styrian theory to use for the purpose of a defence. In the case of *Reg.* v. *Wooler* (Durham Winter Assizes, 1855), it was actually contemplated, by the late Sergeant Wilkins, to account for the unexplained presence of arsenic in the body of Mrs. Wooler, by reference to the opinions of Johnston and Von Tschudi on arsenic-eating. There was an intention to suggest, on the part of the defence, that this lady had for a long period been in the habit of dosing herself with arsenic, unknown to her friends, for the purpose of improving her personal appearance in the eyes of her husband; that her body had become habituated to it, and that, in fact, she had died only because she had latterly left off the practice; but it was prudently abandoned, on the principle that a bad or inadequate explanation is worse than none. According to the evidence given in the case, symptoms of poisoning by arsenic first showed themselves about six weeks before Mrs. Wooler died; they occurred at intervals with aggravation during this period. She had had no access to arsenic in any shape in the six weeks preceding her death. When she died, arsenic was found in all parts of her body—the result of absorption and deposition. The poison was also eliminated in the urine up to within a few days of her death. All the facts were consistent with its being a case of chronic poisoning with arsenic. There was no reason to believe that the deceased had ever taken it voluntarily, or had had arsenic in her possession.

If the exceptional cases observed in Styria are supposed to prove that in this country arsenic may be taken in large doses with impunity, they would lead to error. Such cases have really no practical bearing in legal medicine. Should the practice of arsenic-eating produce no symptom, then no question of poisoning can arise. Should it produce symptoms of poisoning, then the case would fall within the range of ordinary experience. The alleged immunity of the Styrians from the usual effects of this poison by the habitual use of arsenic may be occasionally quoted to explain the detection of arsenic in a dead body, or a motive for its purchase; but no scientific witness who has seen anything of the operation of arsenic in this country would allow these statements to influence his opinion of its effects on human beings generally.

Should arsenic be found in a dead body in small quantity, and there are no appearances indicative of recent administration, the discovery could not embarrass medical evidence, because arsenic is largely used as a medicine; and unless symptoms of poisoning have manifested themselves during life, and there are appearances in the body indicative of its action, there can be no ground for alleging that a person has died from its effects. If, however, such symptoms and appearances are met with, and the poison is found in the dead body, then, the inference will be that the death of the deceased, whether an arsenicophagist or not, was caused by arsenic. The case will then resolve itself into one of accident from overdose, suicide, or murder; and, as in *Reg.* v. *Wooler*, unless it can be proved clearly and conclusively that some one administered the poison, a charge of murder could not be sustained.

An extraordinary use for the purposes of a defence was made of this Styrian doctrine at the trial of Miss Madeline Smith (Edinburgh Court of Justiciary, July, 1857) for the murder of L'Angelier. To account for the purchase of arsenic, the accused stated that she had used it as a *cosmetic.* The deceased died evidently from the effects of arsenic on March 23. Irrespective of two previous purchases of colored arsenic, for which false reasons had been assigned, it was proved that the prisoner had purchased one ounce, as she said, "to kill rats," on March 18, only five days before the death of the deceased. The arsenic was sold colored with indigo. When charged with the crime, and required to account for the poison, she stated that she had bought arsenic on various occasions; that she had used the whole of it as a cosmetic, and had applied it to her face, neck, and arms, diluted with water; that a companion at school had told her that arsenic was good for the complexion. This was directly contradicted by the person whom she had named as her informant; it was proved that she had left school in 1853, and that her purchases of arsenic for cosmetic purposes had only commenced four years afterwards, in February, 1857, *i. e.*, during her secret intimacy with the deceased. It was urged, that this mode of using arsenic externally had never been suggested in any popular publication. In fact, Von Tschudi has not recommended the use of this mineral for washing the face; and an ounce of arsenic, colored with indigo, could scarcely be expected to improve the complexion. This, however, it was suggested might have arisen from ignorance or mistake on the part of the accused respecting the precise mode of using it. To support this theory, Dr. Laurie was called, and he deposed that he had washed his hands and face in water containing a quantity of arsenic colored with indigo, and he had found no disagreeable effects from it. Soon afterwards however he washed his face with cold water, and he stated that he would not advise the external use of arsenic as a practice (p. 26).

It is hardly a question of science, but one of common-sense, whether a woman of adult age would use an ounce of arsenic colored with indigo or soot in the manner and for the purposes suggested! A physician, knowing the properties of arsenic, would take care to keep the poison out of his eyes, nose, and mouth, and relieve himself of risk by speedy ablution afterwards. It is to be hoped that the evidence of this physician as to the immunity which *he* experienced, will not induce others to improve upon Von Tschudi's practice, and freely use arsenic externally as well as internally for benefiting the complexion.

Tolerance.—There are certain conditions of the body in which, without reference to habit, a large dose of a poisonous substance may be taken at once by a person who may not have previously taken it as a medicine, and yet the ordinary effects of poisoning will not be manifested. In tetanus and hydrophobia, poisonous doses of opium have been given at short intervals without producing any injurious symptoms.

Tartar emetic presents a similar peculiarity. This medicine has been safely and beneficially prescribed in large doses and for a long continuance in pulmonary diseases and rheumatism. Tommasini and Laennec were in the habit of prescribing it largely on the Italian theory of contra-stimulus. Persons affected with pulmonary diseases manifested, generally speaking, a "tolerance" of the medicine, if given in large doses and at short intervals. When this tolerance was once set up, the medicine was productive of benefit, but when not established, either from peculiarity of constitution or other causes, it was withdrawn. (See Pereira, *Materia Medica*, 4th ed., vol. 1, p. 101; Forbes's *Translation of Laennec on Diseases of the Chest*, pp. 251, 260; also *Della Nuova Dottrina Medica Italiana*, del Prof. Giacomo Tommasini, Firenze, 1817.) The facts connected with the tolerance of certain medicines in poisonous doses are of some medico-legal interest. Although well known to professional men, it is remarkable that they should have been actually adduced by learned physicians as furnishing a proof that tartar emetic is not a poison, and is not likely to destroy life!

Idiosyncrasy—Intolerance of Poisons.—Idiosyncrasy is a term applied to a peculiar condition of body in which the action of poisons becomes intensified. Small medicinal doses of opium, arsenic, strychnia, mercury, or antimony, may so seriously affect a person as to endanger life. It is well known that some persons have an intolerance of mercury or opium, and that they suffer severely from the administration of these drugs in any form. Alarming symptoms may be produced, or even life may be destroyed by what may be called a non-fatal or medicinal dose of a substance.

Thus, one-fortieth of a grain of strychnia, which is only half of a medicinal dose for an adult, has produced uncomfortable muscular contractions of a tetanic kind. In another case, one-fiftieth of a grain, used hypodermically for the first time, caused convulsions and insensibility, which continued for several hours. (*Am. Journ. Med. Sci.*, Oct. 1, 1872, p. 387.)

It is probable that some deaths from chloroform may have been due to an intolerance of the vapor when it has been administered carefully in ordinary doses. In nine cases death has taken place within two minutes from the commencement of inhalation. In one case only thirty drops had been taken in vapor, but the patient died in one minute, and in another, so small a quantity as fifteen or twenty drops in vapor proved speedily fatal. These cases may admit of explanation, from the fact that there was in the patients an idiosyncrasy or intolerance of chloroform vapor in small doses. Latent disease of the heart or brain has been supposed to account for the fatal result; but these diseases have not existed in some of the rapidly fatal cases from ordinary doses. As a result of idiosyncrasy, therefore, a common medicinal dose may exert a poisonous, instead of a curative action. On the other hand, irrespective of habit or intolerance from disease, a large dose of a poison may be taken and produce no dangerous consequences. Sir R. Christison mentions a remarkable instance of this kind of idiosyncrasy, in which a gentleman, unaccustomed to the use of opium, took nearly an ounce of good laudanum without any effect. (*On Poisons*, p. 32.) This form of idiosyncrasy by which poisons cease to operate as such, is comparatively rare; but daily experience teaches us that some persons are more powerfully affected than others by ordinary medicinal doses. Some cannot tolerate arsenic or opium in any quantity; others are readily affected with lead-disease from causes from which the greater number of persons do not suffer. There are others on whom small medicinal doses of mercury produce salivation and other serious symptoms. I have known the twenty-fourth part of a grain of tartar emetic to produce in an adult, nausea, vomiting, and extreme depression.

It has been remarked by Dr. Garrod that workers in lead are very prone to gout, and conversely, persons of a gouty habit are very susceptible of chronic poisoning with lead. Dr. Wilks has published several cases of Plumbism illustrative of this statement. (*Guy's Hosp. Reports*, 1870, p. 40.)

There can be no doubt that the effects of arsenical wall-papers are, in a great measure, attributable to idiosyncrasy. The dust which escapes from these papers, and the arsenuretted hydrogen gas which is now proved to be emitted from them (*Pharm. Journ.*, August 1, 1874), are in sufficient quantity to affect a few persons, but the greater number who inhabit these rooms escape. (*Sanitary Record*, July 11, 1874.)

This remark equally applies to workmen in lead factories and to persons engaged in other noxious trades.

A third form of idiosyncrasy is seen where a substance, generally reputed harmless and used as an article of food, produces effects so closely resembling those of poisoning as frequently to have given rise to serious mistakes. This is the case with pork, certain kinds of shell-fish, edible mushrooms, honey, and various fruits. There may be nothing poisonous in the food itself; but it acts as a poison in particular constitutions; whether from its being in these cases a poison *per se*, or rendered so during the process of digestion, it is difficult to say.

The subject of idiosyncrasy is of some importance in a medico-legal view, when symptoms resembling those of poisoning follow a meal consisting of a particular kind of food. In such a case, without a knowledge of this peculiar condition, we might hastily attribute to poison in the food, effects which were due to idiosyncrasy in one or two persons who may suffer. On the other hand, when the effects are really due to poison, we may attribute them to some other cause because the quantity was too small to affect the greater number of those who have been exposed to the same influence.

CHAPTER X.

CLASSIFICATION OF POISONS—SPECIAL CHARACTERS OF IRRITANTS—IRRITANT AND CORROSIVE POISONS—NEUROTIC POISONS—CEREBRAL (NARCOTIC) POISONS—SPINAL—CEREBRO-SPINAL (NARCOTICO-IRRITANT) POISONS—CEREBRO-CARDIAC POISONS.

POISONS were formerly arranged in three classes according to the kingdom from which they were obtained; and thus we had mineral, animal, and vegetable poisons. The inutility of such a classification must be apparent when it is considered that we do not, by adopting it, acquire any knowledge of the properties of a poison or of its action on the body. If applied at all, it should be only in a form subordinate to a physiological classification, so as to allow of an arrangement of poisons in analogous groups. All classifications must necessarily be more or less arbitrary. While poisons do not admit of a perfect arrangement, either according to their effects, the organs which are affected by them, or the kingdom of nature from which they are derived, there is room for the adoption of a modification of these arrangements, which, although not free from objection, appears to me sufficient for practical purposes.

The substances called IRRITANT poisons are so well marked in their characters, that they are retained as a class, divisible into three sections, according to their nature, namely, MINERAL, VEGETABLE, and ANIMAL; and the mineral or inorganic irritants are again divided into four sub-sections. The irritants which are derived from the vegetable and animal kingdoms, excepting savin and cantharides, are not often employed criminally.

The remaining substances ranked as poisons are derived chiefly from the vegetable kingdom. The symptoms which they produce indicate an action on the nervous system and sometimes on the heart. Owing to their effects being chiefly manifested on the brain, spinal marrow, and nerves, they are called NEUROTIC poisons. In the vegetable state, as in the form of roots, leaves, or seeds, they often give rise to pain and irritation in the stomach and bowels, but the active principle or alkaloid when separated from the plant does not produce these effects unless it is of an acrid or corrosive nature.

The large class of NEUROTICS here constructed out of the narcotic and narcotico-irritant classes of Orfila admits of a subdivision into four distinct sections according to the organ or organs specially affected by the poison:

IRRITANT.
- MINERAL.
  - Acid poisons.
  - Alkaline poisons.
  - Non-metallic.
  - Metallic.
- VEGETABLE.
- ANIMAL.

NEUROTICS.
- CEREBRAL.
- SPINAL.
- CEREBRO-SPINAL.
- CEREBRO-CARDIAC.

The *Cerebral* poisons include the pure narcotics, such as opium, with its alkaloid morphia, hyoscyamus, and a few other substances. Their action is, as the name is intended to imply, chiefly confined to the *brain.* They produce stupor and insensibility without convulsions. The *Spinal* poisons are those, the action of which is chiefly confined to the spinal marrow, manifested by violent convulsions, sometimes of the tetanic, and of others of the clonic kind. Spinal poisons do not necessarily cause a loss of sensibility or consciousness; there is rarely any symptom of narcotism when they are taken or administered as poisons. Nux vomica and its alkaloid strychnia are types of this form of poisoning. The *Cerebro-spinal* poisons include those which produce symptoms indicative of an action on the brain and spinal marrow; delirium, convulsions, coma, and paralysis. The serpent-poison is of this nature. Among alkaloids, conia, aconitina, and atropia, as well as the plants from which they are derived, furnish examples of this group. In some instances their effects are specially manifested on the nerves of motion by exciting or paralyzing them, in other instances on the nerves of sensation by exalting or destroying sensibility; but in the greater number of cases, effects are produced on both. Some of the neurotic poisons manifest so decided an action upon the heart that there seems good reason for admitting a subdivision of *Cerebro-cardiac* poisons. The Calabar bean, foxglove, and tobacco are the principal members of this group, but to these may be added the poison of the cobra, antiarine, and the vegetable poisons of some savage tribes.

Irritant Poisons.—The *irritants* are possessed of these common characters; when taken in ordinary doses, they occasion speedily violent vomiting and purging. These symptoms are either preceded, accompanied, or followed by intense pain in the abdomen, commencing in the region of the stomach. The peculiar effects of the poison are chiefly manifested on the stomach and intestines, which, as their name implies, they irritate and inflame. Many substances belonging to this class of poisons possess corrosive properties, such as the strong mineral acids, caustic alkalies, bromine, corrosive sublimate, and others. These, in the act of swallowing, are commonly accompanied by an acrid or burning taste, extending from the mouth down the œsophagus to the stomach. Some irritants do not possess any corrosive action,—of which we have examples in arsenic, the poisonous salts of barium, carbonate of lead, cantharides, etc., and these are often called pure irritants. They exert no chemical action on the tissues with which they come in contact; they simply irritate and inflame them.

Difference between Corrosive and Irritant Poisons.—There is this difference between *Corrosive* and *Irritant* poisons. Under the action of corrosive poisons, the symptoms are commonly manifested immediately, because mere contact produces the destruction of a part, usually indicated by some well-marked symptoms. In the action of the purely irritant poisons, the symptoms are more slowly manifested, rarely showing themselves until at least half an hour has elapsed from the time of swallowing the substance. Of course, there are exceptions to this remark; for sometimes irritants act speedily, though seldom with the rapidity of corrosive poisons. It is important, in a practical view, to distinguish whether in an unknown case, the poison which a person, requiring immediate treatment, may have swallowed, is of an irritant or corrosive nature. This may be generally determined by a knowledge of the time at which the symptoms first appeared after the suspected substance was taken. In this way we may often easily distinguish between a case of poisoning from arsenic and one from corrosive sublimate. There is also another point which may be noticed. As the corrosion is due to a decided chemical action, so an examination of the mouth and fauces may enable us to determine the nature of the poison swallowed.

It has been already stated that there are some irritant poisons which have no corrosive properties, and therefore never act as corrosives; but it must be remembered that every corrosive may act as an irritant. Thus the action of corrosive sublimate is that of an irritant poison, as while it destroys some parts of the coats of the stomach and intestines, it irritates and inflames others. So again most corrosive poisons may lose their corrosive properties by dilution with water, and then they act simply as irritants. This is the case with the mineral acids and bromine. In some instances, it is not easy to say whether an irritant poison possesses corrosive properties or not. Thus oxalic acid acts immediately, and blanches and softens the mucous membrane of the mouth and fauces, but I have not met with any decided marks of what could be called chemical corrosion produced by it in the stomach or viscera. Irritant poisons for the most part belong to the mineral kingdom. There are a few derived from the animal and vegetable kingdoms, but these, if we except cantharides and savin, are not often employed criminally. Some of the gases likewise belong to the class of Irritants.

Neurotic Poisons.—Neurotic poisons act chiefly on the brain, spinal marrow, and nerves. Either immediately or some time after the poison has been swallowed, the patient suffers from headache, giddiness, paralysis, stupor, delirium, insensibility, and in some instances convulsions. The cerebral poisons are those which affect the brain only, have no acrid burning taste like the corrosive irritants; they rarely give rise to vomiting or purging. When these symptoms follow the introduction of the poison into the stomach, the effect may be ascribed either to the quantity in which the poison has been taken and the mechanical distension of the stomach thereby produced, or to the poison being combined with some irritating substance, such as alcohol. The pure cerebral and spinal poisons are not found to irritate or inflame the stomach or bowels.

Notwithstanding the well-defined boundary thus apparently existing between these two classes of poisons, it must not be supposed that each class of bodies will always act in the manner indicated. Some irritants have been observed to affect the brain or the spinal marrow remotely, *i. e.*, through the circulation, and as the result of absorption. This is the case with oxalic acid and arsenic. Both of these common poisons have in some instances, from the first, given rise to symptoms closely resembling those of narcotic poisoning, namely, coma, paralysis, and tetanic convulsions. In a case of poisoning with arsenic which occurred to Dr. Morehead, of Bombay, the symptoms of narcotism were so strongly marked that it was believed at first the man had taken a narcotic. (*Med. Gaz.*, vol. 43, p. 1055.) I have met with one case of poisoning with arsenic in which there was paralysis of the extremities, with an entire absence of purging, during the eight days which the person survived. In fact, there is in some cases a nearly complete substitution of one set of symptoms for another. An intelligent writer has assumed that these unusual effects of irritant poisons are only observed in the final stage, *i. e.*, immediately preceding death; and as these effects are similar in many cases, though produced by different agents, he considers it to be an error on the part of toxicologists to apply the term narcotic to the effects produced by oxalic acid or arsenic. (Billing's *Principles of Medicine*, 107.) The case by Dr. Morehead above quoted shows, among numerous other examples, that narcotic symptoms may be produced primarily by arsenic, and not merely as a secondary result, from exhaustion of the vital powers in the last stage of poisoning. On the other hand, in a case of poisoning with a large dose of opium, there was an absence of the usual symptoms of cerebral disturbance, and the presence of others resembling those of irritant poisoning, namely, pain and vomiting. These are to be regarded as exceptional cases, but they show that we cannot always trust to the symptoms as evidence of the kind of poison taken.

Among the alkaloids classed as neurotics, some manifest symptoms resembling those caused by irritants. They are, in fact, more or less irritants. Dr. Burman found in experimenting with conia, that in cases in which it had a rapidly fatal effect on dogs and cats, it caused excoriation of the tongue, frothing and foaming at the mouth, as well as vomiting. In addition to these symptoms, it has caused much gastro-intestinal irritation. (*On Conia,* 1872, p. 2.)

Serpent-poison, although essentially a neurotic, entering the body by a wound, exerts an irritant action on the parts with which it comes in contact. Besides inflammation and swelling of the wounded parts, sloughing may take place and the person die from septicæmia.

Carbolic acid acts on the brain, producing speedily insensibility; at the same time it is a corrosive and irritant poison in its action on the stomach and bowels. Aconite is one of those poisons marked by a variety of action. It may produce the effects of an irritant on the stomach and bowels; it affects the brain and spinal marrow, causing paralysis; and sometimes its action is directed to the heart, and it then destroys life by syncope.

There is considerable difficulty in making a correct classification of neurotic poisons. With respect to some, their mode of action has not been sufficiently investigated; with respect to others, physiologists are not agreed upon the organ which is specially affected. The results of experiments on animals have differed. *Experimentum fallax judicium difficile.* In explanation of this it may be observed that the action of a poison differs according to the dose and other circumstances. There is a maximum as well as a minimum fatal dose, and these act differently. Thus, tobacco in small doses operates on the muscles, destroying all contractility. The alkaloid of tobacco, nicotina, operates fatally on the brain and spinal marrow—the great nerve-centres—in a few minutes. Physostigma (Calabar bean) in a maximum dose has been found to act upon the heart and cause death by cardiac syncope; while in a minimum fatal dose, the pulsations of the heart are only diminished in frequency, and as the circulation continues, the spinal cord is more and more affected until its function is destroyed and asphyxia caused. (*Fraser, Op. cit.,* p. 28.)

The greater number of the neurotics are derived from the vegetable kingdom. These poisons, when taken in the form of leaves, roots, or seeds, have a compound action, from which they have received the name of *Narcotico-irritants.* At variable periods after being swallowed, they may cause pain, vomiting, and sometimes purging, like irritants; they sooner or later produce delirium, stupor, coma, paralysis, and convulsions, owing to their effect on the brain and spinal marrow; but they vary much in their mode of operation. They possess the property, like irritants, of irritating and inflaming the stomach and bowels. As familiar examples, we may point to hemlock, monkshood, and belladonna. This section of poisons (cerebro-spinal) is very numerous, embracing a large variety of well-known vegetable substances; but they rarely form a subject of difficulty to a medical practitioner. The fact of the symptoms occurring after a meal at which some suspicious vegetables have been eaten, coupled with the nature of the symptoms themselves, will commonly indicate the class to which the poison belongs. Some neurotic poisons have a hot acrid taste, others, such as the aconite or monkshood, produce a numbing or tingling sensation in the lips, while others again are intensely bitter, such as nux vomica, picrotoxia, strychnia, and brucia. A few poisons appear to affect the heart as well as the brain. These have been placed in a group under the name of cerebro-cardiac poisons.

The greater number belong to the class of irritants and to the cerebro-spinal subdivision of the class of neurotics. It is, in fact, rare to find that the brain is affected without the spinal marrow, or *vice versâ*. Hence the number of poisonous substances, which can be truly called cerebral or spinal, are very few.

Among the poisonous gases some act as irritants on the throat and lungs (ammonia and nitrous acid); others act on the brain chiefly (nitrous oxide, carbonic acid, and carbonic oxide), they are cerebral poisons; while others again produce their effects on the brain and spinal marrow, causing coma and convulsions (sulphuretted hydrogen and cyanogen).

We are at present hardly acquainted with the special action of some of the substances enumerated and classified as poisons; they have been arranged in this work according to their effects, as ascertained by toxicologists from experiments on animals, as well as from the few cases in which they have acted as poisons in the human body.

CHAPTER XI.

EVIDENCE OF POISONING IN THE LIVING BODY—SYMPTOMS OCCUR SUDDENLY IN HEALTH—INFLUENCE OF SLEEP—OF INTOXICATION—OF DISEASE—ACTION AGGRAVATED BY DISEASE.

We now proceed to consider the evidence of poisoning in the living body. To the practitioner the diagnosis of a case of poisoning is of great importance, as by mistaking the symptoms produced by a poison, for those arising from natural disease, he may omit to employ the remedial measures which have been found efficacious in counteracting its effects, and thus lead to the certain death of a patient. To a medical jurist a correct knowledge of the symptoms furnishes the chief evidence of poisoning, in those cases in which persons are charged with the criminal administration of poison with intent to murder, but from the effects of which the patient ultimately recovers. The symptoms produced during life constitute also an important part of evidence, in those instances in which the poison proves fatal. At present, however, we will suppose the case to be that poison has been taken and the patient survives. Most toxicological writers have laid down certain characters whereby it is said symptoms of poisoning may be distinguished from those of disease.

1. *In poisoning, the symptoms appear suddenly, while the person is in health.*—It is the common character of most poisons, when taken in the large doses in which they are usually administered with criminal intent, to produce serious symptoms either immediately or within a short period after they have been swallowed. Their operation, under such circum-

stances, cannot be suspended and then manifest itself after an indefinite interval; although this was formerly a matter of universal belief, and gave rise to many absurd accounts of what was termed *slow* poisoning. In modern times, the negroes of Martinique have been said to possess this art, but the researches of Dr. Rufz show that this is an erroneous statement. (*Ann. d'Hyg.*, 1844, vol. 1, p. 392; also vol. 2, p. 170.) It is very true that these powerful agents, given at intervals in small doses, do not cause those striking symptoms upon which a practitioner commonly relies as evidence of poisoning. They may then produce disorder, but of so slight a nature as scarcely to excite suspicion. In fact, under these circumstances, the symptoms often so closely resemble those of disease, that an experienced practitioner may be easily mistaken respecting their origin, especially when no moral circumstances exist to create the least suspicion of criminality on the part of those who are around the patient. Arsenic given in small doses, at long intervals, has thus occasioned symptoms resembling those which depend on chronic disease of the stomach. After repeated attacks and recoveries suspicion may be completely disarmed. Among several cases of this kind which have been referred to me for investigation, was one in which it was alleged that a farmer in one of the midland counties had been poisoned two years before by his housekeeper, who was a respectable person, and most attentive to him as a nurse during his illness. He had been attacked at intervals with vomiting and other signs of disorder of the stomach about three months before his death, but recovered under medical treatment. About eight days before his death the symptoms recurred with greater violence than ever, and he sank under them. They were referred to ulceration of the stomach, so closely did they resemble those of disease. As there was no suspicion of poison, the body was not examined; and nothing would have been known respecting the real cause of death, but for a statement made two years afterwards, by the housekeeper, that she had on two occasions administered to her master small doses of arsenic, and the last, probably from its being larger than the first, had occasioned death. In *Reg.* v. *Wooler* (Durham Winter Assizes, 1855), it was proved that the deceased had been laboring under symptoms of poisoning with arsenic, for a period of about six weeks before her death. The symptoms showed that she must have received the poison at different times in small doses. At first they were referred to disease. It was, however, their continuance and their occasional violent recurrence in spite of treatment, that induced a suspicion of poisoning, which was confirmed by a chemical examination of the urine, and subsequently of the body. This is the only form of slow poisoning now known to toxicologists. Again, there are what are called *accumulative* poisons—substances which, in small doses, given at long intervals, produce scarcely any perceptible effect on the system; but which appear to accumulate in the body, and their power is said to be unexpectedly manifested with sudden and violent energy. To these forms of poisoning, which it is extremely rare to meet with on criminal charges, the characters about to be described are not applicable.

When poison is criminally administered, it is almost always in such doses as to cause the symptoms to appear *suddenly*, and to run their course with great rapidity. The symptoms of poisoning with nicotina, prussic acid, oxalic acid, or the salts of strychnia, generally appear either immediately, or within a very few minutes after the poison has been swallowed. In one case, however, where the dose of prussic acid was small and insufficient to produce death, the poison was supposed by the patient not to have begun to act until after the lapse of fifteen minutes. (*Ed. Med. and Surg. Journal*, vol. 69, p. 72.) The symptoms caused by arsenic and other irritants, and, indeed, by all poisons generally, are commonly manifested in from half an hour to an hour. It is rare that the appearance of the symptoms is protracted for two hours, except under certain peculiar states of the system. Some neurotic poisons, such as the poisonous mushrooms, may remain in the stomach twelve or twenty-four hours without giving rise to symptoms; and this is also affirmed to be the case with some animal irritants, such as decayed meat; but with regard to mushrooms, it has been shown by Dr. Peddie that they have produced symptoms in half an hour; and a case has fallen under my own observation, in which the symptoms from noxious food came on within as short a time after a meal as is commonly observed in irritant poisoning by mineral substances. In some cases of poisoning by phosphorus and nitrobenzole, no symptoms have appeared until after the lapse of several hours. These poisons are readily recognized by their odor.

Influence of Sleep.—The symptoms produced by some of the more common poisons are apt to be retarded under certain conditions of the system. When an irritant poison is taken on a full stomach, the symptoms do not usually appear so speedily as when the stomach is empty. So again, it is stated by Sir R. Christison, from cases which have fallen under his notice, that *sleep* retards the action of arsenic, and the same may hold with other poisons. Dr. St. Clair Gray has collected several cases of poisoning with strychnia in which the persons had fallen asleep soon after taking the poison in the form of pills, and the appearance of the symptoms was materially delayed. A boy æt. 12, took three grains of strychnia in a hard pill. He fell asleep and remained so for two hours and a half, when he awoke screaming in a tetanic spasm. A medical man swallowed three grains of strychnia and afterwards fell asleep. He remained so for an hour and a half, awaking with loud cries in a tetanic spasm. (*On Strychnia*, p. 37.) J. P. Cook, after taking two pills given to him by W. Palmer, fell asleep. He awoke with a scream an hour and a quarter afterwards. Some experts at the trial of Palmer, founding their opinion on experiments on dogs and cats, deposed that strychnia could not have been the cause of death in Cook, because the interval for the recurrence of symptoms was too long! In a future case, this matter will be better understood, and due allowance made for the effect of sleep. In reference to irritants some hours may elapse before symptoms appear, and with respect to the alkaloids, one, two, or three hours may pass before the symptoms commence. This influence is supposed to be owing to the general state of insensibility of the body, and the depressed condition of the nervous system during sleep.

Influence of Intoxication.—This state has been considered to retard the operation of opium and other narcotics. Observations of this kind must, of course, be accidental, and there is scarcely a sufficient number of cases reported of narcotic poisoning under these circumstances, to justify a decided opinion on the point. It was observed of a person who had swallowed a strong dose of opium, while partially intoxicated, that the symptoms were some hours before they were manifested. Perhaps, strictly speaking, the symptoms in these cases are masked.

Influence of Disease.—A *diseased* state of the body may render a person comparatively unsusceptible of the action of some poisons, while in other instances it may increase their action, and render them fatal in small doses. In dysentery and tetanus, a person will take, without being materially affected, a quantity of opium sufficient to kill an adult in average health. Mania, cholera, hysteria, and delirium tremens are also diseases in which large doses of opium may be borne with comparative impunity. In a case of hemiplegia, a woman æt. 29, took for six days three grains of strychnia daily without injurious consequences—the dose having been gradually raised (*Gaz. Med.*, Mai, 1845); while one grain of strychnia is commonly regarded as a fatal dose to a healthy person. In a case of tetanus, Dupuytren gave as much as two ounces of opium at a dose (60 grammes), without serious consequences. (Flandin, *Traité des Poisons*, vol. 1, p. 231.) It has also been remarked that persons affected with tetanus are not easily salivated by mercury. (*Colles's Lectures*, vol. 1, p. 77.) The effect of certain diseases of the nervous system as well as of habit, either in retarding the appearance of symptoms, or by *tolerance*, in blunting the operation of a poison, is well known; they are cases which can present no practical difficulty to a medical jurist.

On the other hand, in certain diseased states of the system, there is an increased susceptibility of the action of poison, or what is termed *intolerance* of certain drugs. Ordinary medicinal doses may in such cases exert a poisonous action. Thus, in persons who have a tendency to apoplexy, a small dose of opium may act more quickly and prove fatal. In one laboring under inflammation of the stomach or bowels, there would be an increased susceptibility of the action of arsenic or other irritants. In cases of debility from any cause, these mineral substances would also act injuriously even in ordinary doses. Antimony is a most powerful depressant, and in a small dose it might, by its effect on a diseased heart, cause sudden death by syncope. The influence of disease in increasing the operation of poison, has been noticed in cases of diseased kidney (granular degeneration), in which small doses of mercury have produced severe salivation, leading to exhaustion and death. (*Guy's Hosp. Rep.*, Oct. 1846, p. 443.) In diseases of the lungs affecting aged persons, opium, in medicinal doses, has been observed to exert a poisonous action. The effect of the drug appears to be intensified by the disease. This observation applies equally to morphia. Chloroform vapor in ordinary quantity has been found to produce fatal effects in cases in which there was latent disease of the heart or of the coronary arteries of this organ. A fatty condition of the muscular tissue, leading to great feebleness of the heart's action, appears to be highly favorable to death by syncope under the use of chloroform. A knowledge of these facts is of importance in reference to charges of malapraxis, when death has arisen from ordinary or extraordinary doses of medicines, administered to persons laboring under disease. In such cases, another mode of treatment should be substituted, or a smaller dose than usual given, and its effects carefully watched. In some instances, however, full and large doses of powerful drugs have been recklessly given, and when a fatal result has followed, there has been a strong disposition to refer death to idiosyncrasy, or to the supposed disease, of which, however, sometimes no trace could be found in the body. An experienced physician, well acquainted with pathological anatomy, informs me that, since the use of chloroform has become general, and deaths under its use are not unfrequent, a fattiness and flabbiness of the muscular structure of the heart have been sought for, and almost invariably found! The fatal result has not been attributed to its real cause, the imprudent or careless administration of chloroform, but to some minute structural changes revealed by the microscope in the substance of the organ.

Symptoms appear during a state of Health.—Symptoms of poisoning may manifest themselves in a person while in a state of *perfect health*, without any apparent cause. This rule is of course open to numerous exceptions, because the person on whose life an attempt has been made may be actually laboring under disease; and, under these circumstances, the symptoms may be so obscure as often to disarm all suspicion. When poison is secretly given in medicine, a practitioner is very liable to be deceived, especially if the disease under which the person is laboring, is of an acute nature, and is attended with symptoms of disorder in the stomach and bowels. Several cases of poisoning have occurred in which arsenic was criminally substituted for medicine, and given to the patients while laboring under disorder of the bowels. We are, however, justified in saying, with respect to this character of poisoning, that when, in a previously healthy person, violent vomiting and purging occur suddenly and without any assignable cause, such as disease, indiscretion in diet, or pregnancy, to account for them, there is strong reason to suspect that irritant poison has been taken. When a person is already laboring under disease, we must be especially watchful of the occurrence of any sudden change in the character or violence of the symptoms, unless such change can be easily accounted for on common or well-known medical principles. In most cases of criminal poisoning we meet with alarming symptoms without any obvious or sufficient natural cause to explain them. The practitioner will of course be aware that there are certain diseases which are liable to occur suddenly in healthy people, the exact cause of which may not at first sight be apparent; therefore this criterion is only one out of many on which a medical opinion should be founded.

As a general principle, it may be affirmed that, whenever the body is much debilitated by disease, poisons acquire greater virulence of action. These facts connected with the influence of disease are obviously of some importance in relation to those cases where the person who has taken the poison is already in a diseased or exhausted state.

Thus, then, there are but few exceptions to the rule laid down, that the symptoms of poisoning are liable to appear suddenly; and that in most cases they are manifested within an hour after the substance has been taken.

It has been said that the symptoms of poisoning are characterized either by a *regularity* of increase, or by their becoming more and more aggravated as the case advances; but this is a weak criterion. In the operation of most of the active irritants, there are often remissions, and occasionally intermissions of the symptoms, so as to give rise to false hopes of recovery. It must not, therefore, be inferred that a recurrence of the symptoms of irritation is necessarily indicative of the administration of a fresh dose of poison. The character of the symptoms is in some cases liable to be suddenly changed: vomiting may cease, and may be succeeded by coma. While, then, on the one hand, such a case might, by our trusting too much to this criterion, be regarded as one rather of disease than poisoning, there are, on the other hand, certain diseases which are very rapid and violent in their progress; and the symptoms of these might, for a similar reason, be mistaken for those of poisoning.

The observations here made chiefly refer to irritant poisoning; but they apply with equal force to the administration of neurotic poisons. If a person in health is suddenly seized with stupor, convulsions, delirium, or insensibility, we have just ground for suspicion, unless some natural cause be apparent. Many forms of nervous disease may attack a person in health suddenly, and therefore a careful observation of the symptoms should be made in reference to their mode of commencement, nature, progress, duration, amenability to treatment and result.

CHAPTER XII.

EVIDENCE OF POISONING—SYMPTOMS CONNECTED WITH FOOD OR MEDICINE—SUDDEN DEATH FROM NATURAL CAUSES MISTAKEN FOR POISONING—SEVERAL PERSONS ATTACKED SIMULTANEOUSLY—EVIDENCE FROM THE DETECTION OF POISON IN FOOD AND URINE—MEDICAL DIAGNOSIS IN SECRET POISONING—WHAT TO OBSERVE IN CASES OF SUSPECTED POISONING.

2. *In Poisoning, the Symptoms appear soon after a Meal, or soon after some solid or liquid has been taken.*—This is by far the most important character of poisoning in the living body. It has been already stated that most poisons begin to operate within about an hour after they have been swallowed; and although there are a few exceptions to this remark, yet they occur under circumstances easily to be appreciated by a practitioner. Thus, then, it follows that, supposing the symptoms under which a person is laboring to depend on poison, the substance has most probably been swallowed, either in food or medicine, from half an hour to an hour previously. It must be observed, however, that cases may occur in which the poison has not been introduced by the mouth. Oil

of vitriol and other corrosive liquids have been thrown up the rectum in injections, and have thus caused death; the external application of arsenic, corrosive sublimate, and cantharides to ulcerated surfaces has destroyed life. In one case arsenic was introduced into the vagina of a woman, and she died in five days under all the symptoms of arsenical poisoning. (Schneider, *Ann. der ges. Staatsarzneikunde*, vol. 1, p. 229.) In another instance (*Reg.* v. *Wooler*) there was reason to believe that arsenic had been administered to the deceased in an enema. Such cases are rare, but, nevertheless, the certainty that they have occurred, where their occurrence could hardly have been anticipated, shows that in a suspicious case a medical man must not deny the fact of poisoning merely because it may be proved that the person could not have taken the poison in the usual way, by the mouth. Again, persons may be destroyed by the vapors of ether, chloroform, prussic acid, or other powerful volatile poisons, introduced into the body through the lungs. Such a mode of suicide, or murder, might disarm suspicion, from the fact of no noxious material being found in the stomach.

Let us suppose, however, the circumstances to have been such that these secret means of destruction could not have been resorted to, and that the poison is one of those most commonly selected by a murderer, such as arsenic, tartar emetic, or corrosive sublimate, then we may expect that this character of poisoning will be made evident to us, and that something must have been *swallowed* by the patient shortly before the alarming symptoms appeared. By observations attentively made, it may be in our power to connect the appearance of the symptoms with the use of a particular article of food, and thus indirectly lead to the detection of a criminal. Supposing that many hours have passed since food or medicine was taken by the patient, without any effect ensuing—it is probable that the symptoms may be due to natural causes, and not to poison. When symptoms resembling those of poisoning speedily follow the ingestion of food or medicine, there is, however, reasonable ground for suspicion; but caution should be observed in drawing inferences, since the most extraordinary coincidences sometimes present themselves. The time of the recurrence of symptoms in relation to a particular meal, is then a fact of especial importance in forming an opinion in a case of suspected poisoning.

The Crown Prince of Sweden was considered by many to have been killed by poison. The prince, it appears, was reviewing some troops at Stockholm, in May, 1810, when he was observed to fall suddenly from his horse, and he died in half an hour afterwards. His physician, Dr. Rossi, was accused of having administered poison to him, and he was obliged for his own security to quit the country. It is obvious, however, from an examination of the particulars of the case, that had this sudden attack been due to poison, it could have been only one of most active narcotics, given to the deceased but a short time before he fell from his horse. But it was ascertained that the prince had taken neither solid nor liquid of any kind for at least *four hours* previously to his death. The allegation of poisoning was thus disproved, for no poison, operating with symptoms like those under which the prince had died, could have had its effects suspended for four hours. The cause of death was apoplexy.

Cases of this kind are most instructive to the medical jurist. They show that under a proper observation of the facts, it will not be difficult to distinguish disease from poisoning as a cause of sudden death. They will enable him to do much more than this. In a case of imputed poisoning, he may have it in his power to establish the innocence of one who is accused, or to point out the guilty person when more than one may be suspected. The subjoined cases will serve as additional illustrations of the application of these principles in the diagnosis of poisoning. It is the more necessary to attend to them because there is rather a prejudice among medical men which leads them to suspect death from poison if they cannot at once refer it to any well-known disease.

A woman accused her husband of having attempted to poison her. She handed to the authorities a dish containing arsenic in coarse powder, and some food which she stated had been prepared for her by her husband. On analysis, this was found to contain a large quantity of arsenic. The husband was immediately committed to prison. The wife was at this time apparently quite well, and so she remained for *eight days* afterwards, no symptoms of poisoning having manifested themselves. She was then seized with a fit of mania, and died the following day, *i. e.*, nine days after she had accused her husband of having administered arsenic to her in her food. On examination of her body, it was evident she had died from the effects of arsenic. This poison was found in large quantity in the alimentary canal; and there were the usual morbid changes in the stomach and intestines. The man denied that he had at any time administered poison to the deceased. This denial, however, would have availed him but little, had it not been for the careful medico-legal investigation of the whole case, made by the medical witnesses. As he had been confined in prison *eight days* before the death of his wife, he could not have committed the crime imputed to him, unless he had administered the arsenic previous to his imprisonment. His guilt, therefore, rested upon the medical question whether a large quantity of arsenic could be taken by a person and remain in the stomach and intestines, without producing any of its usual effects, for the long period of *eight days?* The medical experts very properly answered the question in the negative, and the man was immediately discharged. (*Annales d'Hygiène*, 1836, vol. 2, p. 391.) While the prisoner was with his wife she did not suffer from the symptoms of poisoning, nor was there any proof that he had admininistered poison. When, however, he was so situated that he could not possibly have been accessory to its administration, she had suffered from the symptoms and had died from the effects of arsenic. It was fortunate for the accused that he was thrown into prison, and that the case fell into the hands of persons versed in the subject of legal medicine. The interval (eight days) was here too long to be consistent with the theory that he had administered the arsenic found in the body of the deceased.

In the following case the poison selected by the criminal was one in which the symptoms are produced immediately, or within a few minutes, a fact which mainly led to the conviction of the accused:

Jean Humphreys was tried at the Aberdeen September Circuit, 1830, for the murder of her husband, by pouring sulphuric acid down his throat as he lay asleep in bed. The parties frequently quarrelled, and were both addicted to habits of intoxication. On the night in question, some friends had passed the evening with them, drinking. They left the house about twelve o'clock, and soon after this the deceased was seen asleep in bed. The only persons in the house at this time were the prisoner and a servant-maid, and the street door was locked, so that no other person could have had access. The prisoner left the servant's room on her stocking-soles, a thing unusual for her, and when she returned in about twenty minutes, she told the servant that her husband was roaring mad with drink. The girl, upon going to him, found him lying upon his back, declaring that he was all roasting. The prisoner at first showed an unwillingness to send for a medical man, but at length did so. When the deceased left his guests at midnight, there were only two glasses on the table in the room; but when the neighbors came in, after the alarm, there were *three*, and the third was proved to have come from a room above stairs, of which the prisoner had the key. This glass contained, it was supposed, sulphuric acid. In the room where the deceased was lying, there was a vial which had contained sulphuric acid, but it was then nearly empty. The deceased lived two days, but never could give any further account of the matter than that he went to sleep quite well, and awoke "all roasting," and had suffered the utmost agony ever since. He evidently died from the effects of sulphuric acid, large quantities of which were detected on his shirt, on the blanket and bedcover, and a little on the prisoner's bedgown and handkerchief; but not a trace of the poison could be discovered in the stomach or intestines of the deceased. (Alison, *Criminal Law of Scotland*, p. 75; also *Med. Gazette*, vol. 8, p. 77.) In the defence, it was alleged that the deceased had voluntarily taken the poison and committed suicide; but the only time at which he could by any possibility have taken it was when he was drinking with his friends; for immediately after they left, he went to bed, and was seen asleep; and, according to the evidence, he awoke suddenly with the pain and other symptoms produced by this poison. It was impossible that he could have swallowed the acid while drinking with his friends; for the symptoms of the corrosives come on *rapidly*, and cannot be suspended; therefore the poison must have been poured down his throat while he was sleeping, and as the house was at that time fastened up, this act could only have been perpetrated either by the prisoner or the maid-servant. There was nothing to throw suspicion on the servant, and all the circumstances pointed to the wife as the guilty person. She was convicted and executed. It will be observed that the suspicion of suicide, as well as of murder by the persons with whom the deceased had been drinking, was entirely removed by attention being paid to this well-marked character of the corrosive poisons.

It has been already stated that, if symptoms resembling those of poisoning speedily follow the introduction of food or medicine into the stomach, there is great room for suspicion; but caution should be observed in expressing an opinion. In the case of Sir Theodosius Boughton, who was poisoned by his brother-in-law, Donellan, in 1781, the fact of alarming symptoms coming on in *two minutes* after the deceased had swallowed what was supposed to be a simple medicinal draught, became a most important piece of evidence against the accused. There is no doubt that laurel water had been substituted for the medicine by the prisoner. (See LAUREL WATER, *post.*) The practice of substituting poisonous mixtures for medicinal draughts or powders, is by no means unusual, although it may be supposed to indicate a degree of refinement and knowledge not commonly to be found in the lower class of criminals. In some cases, poisons may have been ignorantly dispensed for medicine. Medical practitioners may thus be fatally deceived. The late Baron Alderson on one occasion publicly related the following case: An apothecary prepared a draught, into which another person put poison, intending thereby to destroy the life of a patient for whom the medicine was prescribed. The patient, not liking the taste of the draught, and thinking that there was something suspicious about it, sent it back to the apothecary, who, knowing the ingredients of which he had composed it, and wishing to prove that he had done nothing wrong, drank it himself, and died. In this case, he was the unconscious agent of his own death; and although the draught was intended for another, the person who poisoned it was held guilty of murder.

We should remember that on these occasions poison may have been accidentally or criminally substituted for an innocent medicine prescribed. In 1856, a physician of this metropolis nearly lost his life by drinking what he supposed to be an infusion of ash-leaves. He had prescribed this infusion for a patient who had been rendered insensible by the first dose; and, in order to satisfy himself of its nature, he drank only a small quantity. It turned out that an ignorant herb-dealer had sold the leaves of belladonna for those of the ash; it was an infusion of belladonna which had thus been taken by the patient and physician. In a case which was referred to me some years since, a medical man put into a mixture a large quantity of strychnia in place of oxide of bismuth. His patient, a lady, took a dose and died in twenty minutes, with the usual tetanic symptoms. The medical man was sent for and informed of his mistake. He denied that any mistake had been made, and to confirm this denial he swallowed some of the mixture from the mouth of the bottle, but fortunately for himself without shaking it. In half an hour he was seized with symptoms of poisoning with strychnia, and almost succumbed. Although he did not intend it, he thus furnished the strongest evidence against himself. He was tried for manslaughter at the Rutland Assizes and acquitted, on the ground of its having been an innocent mistake!

The occurrence of symptoms resembling those produced by poison, soon after a solid or liquid has been taken, may be a pure coincidence. In such a case, poisoning is always suspected by the vulgar; and it will be the duty of a medical jurist to guard against the encouragement of such a suspicion, until he has strong grounds for believing it to be well founded. No public retractation or apology can ever make amends for the injury which may in this way be inflicted on the character of another; for those who hear the accusation may never hear the defence. In such cases, a practitioner may entertain a suspicion, but, until confirmed by facts, he should always avoid *expressing* it, giving it publicity, or encouraging the expression of it by others. When death is not a consequence, it is difficult to clear up such cases, except by the aid of a chemical analysis; but this, as we know, is not always applicable. If death ensue, the real cause is usually apparent, and a suspicion of poisoning may be thus removed by an examination of the body and an analysis. In some cases, in which persons have died suddenly after a meal, the cause of death has been clearly traced to an obstruction of the air-passages by a mass of food. These parts should always be carefully examined.

An instance occurred within my knowledge, where an aged lady took three grains of a white powder, prescribed for her by her medical attendant. In about ten minutes afterwards she was seized with coma, and died in the course of an hour. The medicine she took was sulphate of quinine. In such a case it might have been most plausibly said morphia or some other poisonous alkaloid had been swallowed; but the circumstances were well known: death was due to apoplexy. In another case, a woman, aged 37, rose in the morning in her usual health, with the exception of having a slight headache; immediately after taking breakfast she was attacked with violent vomiting, which continued for half an hour—she then fell down and died suddenly. Here again there was room for suspecting poison, owing to the time of the occurrence of symptoms, but it was proved that the woman had died from disease of the brain. The fatal symptoms produced by perforation of the stomach, which in some respects resemble those of arsenical poisoning, almost always attack a person soon after a meal. When they occur some hours afterwards, there is less likelihood of confounding them with arsenic. Some years since, Mr. Hilton and myself were required to examine judicially a case of this description. Our judgment was in a great measure aided by the fact that the violent symptoms did not appear until about three hours after a meal.

3. *In Poisoning when several partake at the same time of the same Food or Medicine (mixed with Poison) all suffer from similar Symptoms.*— This character of poisoning cannot always be procured; but it furnishes good evidence of the fact when it exists. Thus, supposing after a meal made by several persons from the same dish, only one suffers, the suspicion of poisoning is considerably weakened. The poisoned article of food may be detected by observing whether they who suffer under symptoms of poisoning have partaken of one particular solid or liquid in common. In a case of accidental poisoning at a dinner-party in London, it was observed that the persons who suffered from the symptoms had taken port wine only. The contents of the bottle were brought to me for examination; they were found to consist of a saturated solution of arsenic in wine. In general, considerable reliance may be placed upon this character, because it is improbable that any common cause of disease should suddenly attack, with violent and

alarming symptoms, several healthy persons at the same time, and within a short period after having partaken of food together. In a case referred to me many years ago, a diabolical attempt to administer poison was shown which might throw a medical man off his guard who relied upon the fact that all who partook of the same meal ought to suffer, supposing the food to be poisoned. A number of persons in a poor-law union sat down to dinner, according to custom, in regular order and in the same places. They were all helped to the food (meat and gravy) in their regular turns. The wife of one of the officials, shortly after commencing her dinner, perceived a white powder floating on the gravy in her plate. Suspecting something wrong, she collected it and put it aside. After her dinner, she was seized with nausea and other symptoms resembling irritant poisoning, but these in a few days passed off, leaving her very weak. The powder was examined, and I found it to be white arsenic. No such appearance presented itself on any other plate, and no other person who dined at the table suffered from symptoms of poisoning. There had been ill-feeling against the woman whose life was thus attempted. There was reason to believe that some person had put the poison into the plate which it was known would come to her in her turn, the plates having been put in a pile before the person who carved the meat. On another occasion, a gamekeeper and his three children sat down to dinner together, and in about half an hour one of the children was seized with vomiting and purging, and died in a few hours. Arsenic was found in the child's stomach, and there was no doubt that this was the cause of death. The man was charged with murder. The great medical difficulty in the case was to explain how it happened, as they all partook of the same food, only one child suffered. An examination of the surviving children brought to light the fact that the only difference made at the dinner was that the father (the prisoner) helped the two survivors to salt from the salt-cellar on the table, but the salt (?) which he put on deceased's plate he brought between his finger and thumb from another room. On a shelf in this room, he kept a bottle of powdered arsenic which he used for destroying vermin. The general and circumstantial evidence clearly showed that the poison had been thus administered to the child. The man was convicted, and executed. (*Reg.* v. *Jennings*, Berks Lent Ass., 1845.)

We must beware of supposing that when poison is placed in the food of which many persons partake all will be attacked with precisely similar symptoms, or at the same interval of time; because, as we have seen, there are many causes which may modify these conditions. In general, that person who has partaken most freely of the poisoned dish will suffer most severely, but even this does not always follow. There is a well-known case recorded by Bonnet, where, among several who partook of a dish poisoned with arsenic, they who had eaten little and *did not vomit*, speedily died; while others, who had partaken largely of the dish, and had in consequence vomited freely, recovered.

It was just now remarked that there is no disease likely to attack several healthy persons at the same time, and in the same manner.

This is undoubtedly true, *as a general principle*, but the following case will show that mistakes may occasionally arise even under these circumstances. It occurred in London, during the prevalence of the malignant cholera in the year 1832. Four of the members of a family living in a state of great domestic unhappiness, sat down to dinner in apparently good health; some time after the meal, the father, mother, and daughter, were suddenly seized with violent vomiting and purging. The stools were tinged with blood, while the blueness of the skin, observed in cases of malignant cholera, was wanting. Two of the persons died. The son, who was known to have borne ill-will against his father and mother, and who suffered no symptoms on this occasion, was accused of having poisoned them. A strict investigation took place before the coroner; but it was clearly shown by the medical attendant that the deceased persons had really died of the malignant cholera, and there was no reason whatever to suspect that any poison had been administered to them. In this instance, it will be perceived that symptoms resembling those of irritant poison appeared suddenly in several persons in perfect health, and shortly after a meal. We hereby learn that the utility of any rules for investigating cases of poisoning, depends entirely on the judgment and discretion with which they are applied to particular cases.

A somewhat similar set of cases appears to have occurred at Vienna, in the summer of 1873, during the prevalence of malignant cholera in that city. The father, mother, and two daughters in a family, while in perfect health, were suddenly seized with incessant vomiting, purging, cramps in the calves of the legs, cold extremities, lividity of the limbs and face, and the pulse was scarcely perceptible. They all recovered. Dr. Rosenthal, who was called to see these persons, pronounced them to be cases of poisoning with Vanilla ice, of which it appears the family had partaken. But Vanilla ice had been frequently eaten in Vienna before the outbreak of cholera in 1873 without such severe symptoms following, and it is highly probable that the medical man was misled by the number of persons who were attacked simultaneously. There is nothing in Vanilla to cause poisoning. It contains a crystallizable principle, Vanillin or Vanillic acid, which is not poisonous. (Husemann, *Pflanzenstoffe*, 1871, p. 1038.) In order to account for the irritant effects, Dr. Rosenthal believes that there was a production of *cardol*, an irritant oily principle (found in Anucardium Occidentale), somewhat resembling cantharidin. The facts are more consistent with the effects produced by malignant cholera. (*Pharm. Jour.*, April, 1874, pp. 838, 852.)

The simultaneous occurrence of symptoms terminating fatally in two or more persons in a family, is always well calculated to excite grave suspicions of poisoning; and a safe opinion can then only be formed by noting the character of the symptoms in each case, or, if this source of evidence be wanting, by the detection of poison in the food or bodies of the individuals. A simultaneous attack merely furnishes a presumption in favor of poisoning, to be supported or rebutted by other circumstances. A case was referred to me in December, 1846, by the late Mr. Wood, coroner for Surrey, in which two children, previously healthy, died under similar symptoms, very suddenly, and after a short illness. It was reasonably suspected, in the first instance, that narcotic poison had been given to them; but an examination of the facts of the case, as well as an analysis of the food and contents of the stomachs, proved that poison was not the cause, and thus removed a heavy load of suspicion from the parents.

Some years since, three children in the family of a medical man in the Isle of Wight, died after a short illness, one after the other, within a week. The symptoms were supposed to bear some resemblance to irritant poisoning. No poison was found in the bodies, and there was no evidence that any poison could have been possibly administered by any member of the household. There was no conceivable motive for such an act. In examining the intestines of one of the children, an infant, I found an intussusception of some extent, and the intestine was partly in a state of strangulation. This was sufficient to account for one death from natural causes, and the other two were coincidental. Had either death taken place by itself, no suspicion of poisoning would have been raised, but because three children had died at or about the same time, poisoning was alleged, and in spite of the negative chemical and the moral evidence, it proved impossible to eradicate that suspicion.

In July, 1874, Mr. Clegg, coroner for Boston, communicated to me a similar set of cases which led to an inquest before him. Three children in the family of a cottager named Slight, died under the following circumstances: On May 18 a girl æt. 5 was suddenly seized with vomiting and purging, and great thirst, and died in the course of a few hours. On May 20, a boy æt. 3 was seized with similar symptoms and died. On May 24, another girl æt. 6 was attacked, and she also died under similar symptoms after an illness of thirty-six hours. The children were not taken ill at the same time but one after the other. They were apparently well, and all of them died and were buried within the short period of eight days. From rumors circulated, affecting the parents, the body of the last girl was exhumed six weeks after death, and examined. The inner coat of the stomach and of portions of the intestines was inflamed. The other organs were healthy. Dr. Stevenson made an analysis of the viscera, but found no poison. The fullest inquiry showed that there was no poison in the house, and no motive for poisoning on the part of the parents. No cause could be discovered to account for the symptoms and deaths of these three children, but it was suspected that they had died from cholera as a result of drinking impure water. When several members of a family are taken ill about the same time, and die about the same time, a suspicion of poisoning may arise; but we should always be prepared to admit that, by a coincidence, a fatal disease may carry off two or more members of a family within a few days of each other.

Obscure symptoms of poisoning may occur simultaneously in several members of a family from accidental causes, the nature of which may not be even suspected. Thus, various articles of food may be poisoned by copper through want of cleanliness in the use of culinary utensils (see COPPER); or the water supplied to a house may be contaminated with lead from the use of that metal in pipes, cisterns, or merely as a cover to a tank (see CARBONATE OF LEAD). The safety of the individuals, and probably the exculpation of an innocent person, wrongly accused of poisoning, will depend on the acumen of the medical attendant in discovering the real cause. It is a mistake to suppose that in this insidious form of poisoning, either all must suffer from the effects, or there is no poisoning at all! Persons exposed to the same influence of chronic poisoning by lead or copper are very differently affected. In the case of the royal family of France, at Claremont, in 1849, although the whole household was supplied with the same water, poisoned with lead, only thirteen out of thirty-eight members of the family suffered from the effects. The cause was in this case, medically speaking, very clearly traced to the water, but in the absence of a proper analysis, it might have been legally pronounced free from poison, because twenty-five persons had escaped.

In July, 1866, a remarkable set of cases occurred in the family of a Mr. Corrie, Itchen Abbas, Hants, in which twelve or more members of the family suffered from symptoms of poisoning similar to those produced by copper in food. A badly tinned copper vessel had been used for cooking the food, with much salt. One patient, an old man, æt. 90, died after three weeks, the others recovered. The cook was charged with wilful poisoning, but was subsequently liberated. She brought an action against her master (*Tully* v. *Corrie*, Queen's Bench, Nov. 1867), but this resulted in a verdict for the defendant. A full account of this case will be found in the *Guy's Hosp. Rep.*, 1866, p. 329.

It may be here proper to remark that the water of wells in the neighborhood of chemical works is often impregnated with poison. Members of a family who unsuspectingly use this water may be attacked with symptoms of poisoning, and die from the effects. In the Registrar-General's Quarterly Report for 1846, it is stated that nearly the whole of the members of a family in Derbyshire died from having drunk water impregnated with arsenic, which was drawn from a well contiguous to certain chemical works attached to the premises. (*Med. Gaz.*, vol. 37, p. 843.)

It is proper to bear in mind, in conducting these inquiries, that symptoms resembling those produced by irritant poison may be occasionally due to the food which may have been taken by a family at a meal. Besides flesh rendered unwholesome from disease and decay, there are certain kinds of shell-fish, muscles and whelks, as well as pork, bacon, sausages, cheese, and bread, which, under certain circumstances, may give rise to serious symptoms, and even death. In such a case, all the foregoing characters of poisoning are brought out; and, indeed, it may be regarded as one of poisoning by an animal or vegetable irritant. These cases present some difficulties; great ambiguity frequently arises from the fact that not more than one or two persons may be affected, who have frequently before partaken of the same kind of food without any particular inconvenience.

4. *The Discovery of Poison in the Food taken, or in the Matters vomited.*—One of the strongest proofs of poisoning in the living subject is the detection of poison by chemical analysis, either in the food taken by the person laboring under its effects, in the matters vomited,

or in the urine, if the poison be one of those which are eliminated by the kidneys. The evidence is, of course, more satisfactory when the substance is discovered in the matters vomited or in the urine, than in the food; because this will show that poison has really been taken, and will at once account for the symptoms. If these sources of evidence are not accessible, then we must examine the food of which the patient may have partaken. Should the results in all cases be negative, it is probable that the symptoms may have been due to disease. In investigating these cases in the living subject, a medical jurist must remember that poisoning is sometimes *feigned*, and at others, *imputed*. It is very easy for an artful person to put poison into food, and to accuse another of having administered it, as well as to introduce it into the matter vomited or discharged from the bowels, or into the urine. There are few of these accusers who go so far as to swallow poison under such circumstances, because there is in general a great dread of poisonous substances; and it will be at once apparent that it would require a person well versed in toxicology, to feign a series of symptoms which would impose upon a practitioner at all acquainted with the subject. In short, the difficulty reduces itself to this: What inference can we draw from the mere chemical detection of poison in food? A medical man may say whether poison is or is not present in a particular article of food; but he must leave it to the authorities of the law to develop the alleged attempt at administration. If the poison should have been actually administered, then we may expect to find the usual symptoms.

With regard to the detection of poison in the matters vomited, this fact affords no decisive proof that the substance has been swallowed, except under two circumstances: 1. When the accuser actually labors under the usual symptoms of poisoning, in which case there can be no feigning, and the question of imputation is a matter to be established by general evidence. 2. When the matters are actually vomited into a *clean vessel* in the presence of the medical attendant himself, or of some person on whose testimony perfect reliance can be placed.

The detection of absorbed poison in the *urine* furnishes in general a clear proof that it has been taken, that it has passed into the blood and has been subsequently eliminated. When the symptoms point to arsenic, antimony, or a mineral poison, this aid to diagnosis should never be neglected. Some years since the following case was referred to me: A lady had suffered from protracted illness attended with occasional vomiting and great depression. From her social position poisoning was not suspected. Still no remedies availed to relieve her symptoms, and she was fast sinking from exhaustion. Various views were taken of the nature of the disease under which it was supposed she was laboring, when the mystery was solved by a portion of the urine being sent to me at Guy's Hospital. Antimony was found in it, and as no antimonial medicine had been prescribed by her medical attendants, there could be no doubt that her symptoms were caused by the secret administration of small doses of tartar emetic by some person in the household. Suspicion fell upon no one, but the report of the analysis was read at the patient's bedside in the presence of all the members of the family, and from that time the symptoms ceased.

In the case of General Ketchum, which was lately the subject of a lengthened trial for murder in America, the contention was on the one side that the General had died from an attack of cerebro-spinal meningitis, and on the other that he had died from the effects of antimonial poison secretly administered to him. It does not appear that any analysis of the urine for poison was made while the deceased was living. This simple proceeding might have spared much conflict of medical opinion.

When a medical man is called to a case of suspected poisoning, it is necessary that he should know to what points he ought to give his attention. It is very proper that every effort should be made by him to save life when the person is living; but while engaged in one duty, it is also in his power to perform another, supposing the case to be one of suspected criminal poisoning, namely, to note down many circumstances which may tend to detect the perpetrator of a crime. There is no person so well fitted to observe these points as a medical man; but it unfortunately happens that many facts, important as evidence, are often overlooked. The necessity for observing and recording them is not, perhaps, generally known. A medical man should not make himself officious on such occasions, but he would be unmindful of his duty as a member of society, if he did not aid the cause of justice by extending his scientific knowledge to the detection of crime. It is much to the credit of the medical profession that the crime of murder by poisoning—a form of death from which no caution or foresight can protect a person—is so frequently brought to light by the announcement of suspicious facts of a medical nature to magistrates and coroners; and on several occasions the highest compliments have been passed by judges on medical men who have been thus indirectly the means of bringing atrocious criminals to the bar of justice.

The following appear to me to be the principal points which demand the attention of a medical jurist in these cases of suspected poisoning: 1. With respect to

Symptoms.—1. The time of their occurrence—their nature. 2. The exact period at which they were observed to take place after a meal, or after food or medicine had been taken. 3. The order of their occurrence. 4. Whether there was any remission or intermission in their progress; or, whether they continued to become more and more aggravated until death, 5. Whether the patient had labored under any previous illness. 6. Whether the symptoms were observed to recur more violently after a particular meal, or after any particular kind of food or medicine. 7. Whether the patient has vomited; the vomited matters, if any (especially those *first* ejected), should be procured; their odor, color, and acid or alkaline reaction noted, as well as their quantity. 8. If none be procurable, and the vomiting has taken place on the dress, furniture, or floor of a room—then a portion of the clothing, sheet, or carpet, may be cut out and reserved for analysis; if the vomiting has occurred on a deal floor, a portion of the wood may be scraped or cut out; or if on a stone pavement, then a clean sponge soaked in distilled water may be used to remove any traces of the substance. The vessels in which vomited matters have been contained will often furnish valuable evidence, since heavy mineral poisons fall to the bottom, or adhere to the sides. 9. Endeavor to ascertain the probable nature of the food or medicine last taken, and the exact *time* at which it was taken. 10. Ascertain the nature of *all* the different articles of food used at a meal. 11. Any suspected articles of food, as well as the vomited matters, should be sealed up as soon as possible in clean glass vessels, labelled and reserved for analysis. 12. Note down, in their own words, all explanations voluntarily made by persons present, or who are supposed to be concerned in the suspected poisoning. 13. Whether more than one person partook of the food or medicine; if so, whether all these persons were affected, and how? 14. Whether the same kind of food or medicine had been taken before or since by the patient or other persons without ill effects following.

CHAPTER XIII.

EVIDENCE FROM THE NATURE OF THE SYMPTOMS—DISEASES RESEMBLING IRRITANT POISONING—CHOLERA—GASTRITIS—ENTERITIS—GASTRO-ENTERITIS—PERITONITIS—GASTRIC FEVER—ULCERATION AND PERFORATION OF THE STOMACH—STRANGULATED HERNIA—INTUSSUSCEPTION—INTERNAL STRANGULATION OF THE INTESTINES—COLIC—HÆMATEMESIS.

Nature of the Symptoms.—The nature and order of occurrence of the symptoms under which a person is laboring, should be accurately observed in a suspected case. In poisoning, the symptoms are commonly well marked, and have a peculiar character; those of disease are less certain, and are more likely to create embarrassment. Owing to this, it happens that, in practice, disease is much more liable to be mistaken for poisoning, than poisoning for disease. An account of the symptoms produced by the two classes of poisons will be found at page 74; and the special details, in the description of each poison respectively. At present it will, therefore, only be necessary to enumerate, on the one hand, those diseases, the symptoms of which might be mistaken for irritant poisoning; and, on the other, those which might be mistaken for neurotic poisoning.

DISEASES RESEMBLING IRRITANT POISONING.

The diseases, the symptoms of which resemble those produced by *irritant* poisons, are cholera, gastritis, enteritis, gastro-enteritis, peritonitis, perforation of the stomach or intestines, strangulated hernia, intussusception, colic, and hæmatemesis.

Cholera.—This name is given to a disease in which there is a combination of vomiting and purging, generally of biliary matter. It is necessary to distinguish the common English cholera from the Asiatic or malignant form of the disease. In the ASIATIC CHOLERA there is usually sudden and extreme prostration of strength; the surface of the body is cold, and it sometimes has a dark livid or leaden hue, especially observed in the skin of the hands and feet. The skin is shrivelled, the features are pinched, and the breath is cold as it issues from the mouth; the matters discharged from the bowels are very copious, resembling rice water with flakes of coagulated mucus floating in them. There is the most intense thirst, and the patient will drink a large quantity of cold water. The symptoms of poisoning with arsenic and other irritants, are wholly different from these, if we except perhaps the intense thirst, which is present in both cases. Dr. Wilkes met with one case of poisoning with arsenic, which proved fatal in nine hours, in which the symptoms were similar to those of malignant cholera. (*Guy's Hosp. Rep.*, 1855, p. 364.)

In poisoning with arsenic the skin is hot and cold at intervals; the pulse frequent, small, and irregular, amounting to from 120 to 130 in a minute. It is only in the last stage of arsenical poisoning (collapse) that there is an icy coldness of the limbs. With the thirst there is commonly great constriction in the throat, not met with in this form of cholera.

The common ENGLISH CHOLERA, as it occurs in summer and autumn, more closely resembles irritant (arsenical) poisoning in its symptoms. Thus an attack often comes on in a healthy person in about half an hour after a meal. It is accompanied by vomiting and purging of bilious liquid, and by violent pain in the abdomen, continuing until death when the case terminates fatally. It may usually be traced to some indigestible food of which the patient has partaken. Many acquittals on criminal charges have taken place from the great difficulty which exists in distinguishing this last-mentioned form of cholera from arsenical poisoning; and, in truth, it may be observed that if in any case medical evidence rested on symptoms alone, it would be scarcely possible, in some instances, to draw such a clear distinction between the symptoms of the disease and those of poisoning, as the law would deem absolutely safe for a conviction on a criminal charge.

The rules recommended for forming an opinion, as they are laid down by the best writers on toxicology, are not satisfactory. Perhaps the following may be taken as a statement of the most striking differences. In irritant poisoning the evacuations are often tinged with blood; in cholera they are not tinged with blood, but commonly deeply colored with bile. In irritant poisoning, these evacuated liquids will sooner or later yield traces of poison when analyzed. In cholera this is of course not the case. The attack of cholera is commonly dependent on some irregularity of diet, and appears chiefly in summer and autumn. Irritant poisoning may occur at any season. Except when it prevails in a severely epidemic form, from intense heat or other causes, and attacks the very aged or the very young, English cholera is not often fatal; and when it does prove fatal, it is commonly after three or four days from its commencement, by exhaustion of the patient. In irritant (arsenical) poisoning, death is a common result in twenty-four hours, when the symptoms produced by the poison are such as to resemble those of cholera, *i. e.* poisoning in its most acute form. In irritant poisoning, the symptoms usually come on in about half an hour or an hour after a meal; and, although cholera may commence its attack at about the same period, yet, supposing several persons to have partaken of the food, all will suffer more or less if it be really a case of poisoning—not if it be a case of cholera. It would be at least something very unusual, that several healthy persons should be attacked by cholera at the same time, unless the attack were owing to some improper kind of food used at the meal. (See p. 88.) Lastly, an analysis of the food or urine may serve to determine whether irritant poison was or was not the cause of the symptoms. Of all irritant poisons, arsenic comes the nearest to cholera in the character of the symptoms. It is right to bear in mind, however, that a case of arsenical poisoning is often accompanied by special symptoms which are met with neither in cholera nor in any disease resembling it. Thus in persons who have taken arsenic and survived the first effects of the poison—the conjunctivæ (whites) of the eyes often become inflamed, sometimes at a very early period—there is also great irritation of the skin, followed by a peculiar (eczematous) eruption; and occasionally numbness, or tingling in the hands and feet, as well as paralysis and coma, appear among the symptoms. In cholera, nothing of this kind is witnessed; hence we have in these peculiar symptoms of arsenical poisoning, means for assisting us in forming an opinion. When the person dies, an examination of the body with an analysis of the contents of the stomach, or if death speedily follows the attack, an analysis of the tissues of the soft organs, will often remove any doubts that may have existed on the real nature of the case. In numerous cases, arsenical poisoning has been mistaken for cholera, and the fact of poisoning has remained concealed until an analysis was made. (See cases of *Reg.* v. *Chesham*, Essex Lent Assizes, 1847; and *Reg.* v. *Foster*, Suffolk Lent Assizes, 1847.) M. Tardieu has fully examined the medico-legal bearings of this subject. (*Ann. d'Hyg.*, 1854, vol. 2, p. 162.)

As *chronic* irritant poisoning bears some resemblance to chronic disease, these cases frequently give rise to a conflict of medical opinions. This may especially occur in reference to chronic poisoning with antimony, which sometimes causes death by syncope from the great exhaustion and depression produced. Many of the symptoms may be consistent with chronic disease of the stomach, irrespective of poisoning. The only safe guide to a proper diagnosis in such cases is a careful chemical analysis of the urine while the person is living, and of the various organs of the body after death. In the case of Ann Palmer, the wife and one of the victims of William Palmer, the symptoms were such as poisoning with antimony would cause: incessant vomiting after taking certain articles of food, bilious purging, great depression, low pulse, and death after a few days from exhaustion. But the symptoms were also such as an attack of English cholera might explain. On an exhumation of the body, a year after death, Dr. Rees and I found the solid sulphide of antimony in the stomach, and antimony was found in the liver and in all the parts examined, even in the ovaries. No antimony had been prescribed for her during her illness. From this discovery we had no hesitation in assigning death to chronic poisoning with some antimonial preparation. After these facts had been made known at the inquest, and a verdict returned accordingly, a respectable physician wrote an elaborate pamphlet to prove that this lady had died, not from chronic poisoning, but from a severe attack of summer cholera, the saturation of the body with antimony being ignored, or treated as an unimportant coincidence!

Gastritis, Enteritis, Gastro-enteritis, Peritonitis.—These diseases do not commonly occur without some obvious cause; indeed, the first two, in the acute form, must be regarded as the direct results of irritant poisoning. Thus arsenic and other irritants, when they prove fatal, commonly give rise to inflammation of the stomach and bowels. In all cases in which these diseases present themselves, the object of a practitioner is therefore to determine the *cause* of the inflammation, whether it be due to natural disease, or the action of an irritant. The distinction will chiefly rest, 1. Upon the time of the occurrence of the symptoms after a meal; 2. The order of their occurrence; 3. The obstinate constipation of the bowels, which is observed in gastritis and enteritis, as contrasted with the violent vomiting and purging met with in irritant poisoning; 4. The presence of fever in these diseases. The history of the case so clearly explains its nature, that we seldom hear of these diseases being mistaken for irritant poisoning. The same observations apply to peritonitis, in which disease there is also constipation, and but little vomiting, with general tenderness over the whole of the abdomen. It has been doubted by some pathologists, whether the diseases abovementioned can occur spontaneously, and without any apparent cause. All agree that instances of idiopathic acute gastritis are rarely observed in persons otherwise healthy. Two cases were reported to the Medico-Chirurgical Society, by Dr. Burne (*Med. Gaz.*, vol. 25, p. 414), and another case has been published by Dr. Berncastle. (*Lancet*, March, 1844.) The symptoms were of the usual character,—constant vomiting, no purging, and rapid sinking. After death the stomach was found in a high state of inflammation, but all the other organs were healthy. A suspicion of poisoning did not attach to the case. Acute enteritis from natural causes is much more common than acute gastritis. These diseases, in a chronic form, have a very slow course, and may be a secondary result of irritant poisoning. The symptoms are unlike those produced in the acute form of poisoning. The case of *Reg.* v. *Hunter* (Liverpool Spring Assizes, 1843) was successfully defended on the theory of gastro-enteritis from natural causes, in spite of the strongest suspicions that arsenic was the cause of death.

Gastric Fever.—It could scarcely be supposed that a case of arsenical poisoning should be mistaken for this disease. The case of Mary Ann Cotton (*Reg.* v. *Cotton*, Durham Lent Assizes, 1873) shows, however, that such a mistake may be made not only once but in a series of suspicious cases. One out of three husbands, and four of her children died rather rapidly one after another, with symptoms of irritant poisoning, proved by the exhumation and examination of the bodies to be owing to arsenic. Gastric fever was certified by the medical man to be the cause of death. Under such a perfunctory mode of registering causes of death it is not surprising that this woman succeeded in destroying by poison twenty persons before her crimes were discovered!

In arsenical poisoning there is no fever, while in the disease the symptoms are chiefly those of fever with vomiting, from the irritable state of the stomach, the vomited matters presenting no marks of blood or poison. In irritant poisoning there is violent and bloody purging. This is not a symptom of gastric fever. There is an absence of the severe burning pain, and after death the appearances in the stomach and intestines are wholly different.

Perforation of the Stomach and Intestines.—The symptoms attending perforation of the stomach in some respects resemble those of irritant poisoning. They often occur suddenly to a healthy person after a meal. This disease is almost invariably fatal, and may be immediately recognized on the examination of the body. Even in the rare cases in which it is not fatal, the means of diagnosis are not difficult. (See *post.*)

Strangulated Hernia.—It is difficult to suppose that this disease should ever be confounded with irritant poisoning. The seat of pain, with an examination of the part, would at once show the physical cause to which the symptoms were due. (See *Ann. d'Hyg.*, 1854, vol. 2, p. 143.)

Intussusception of the Bowels—Ileus, Iliac Passion, Internal Strangulation.—These terms are applied to a disease in which there is violent vomiting without purging; the mechanically locked state of the bowel preventing the passage of fæces, or allowing only blood and mucus to pass. It differs from diarrhœa, in which there is purging without vomiting, and from cholera, in which there are both. In irritant poisoning, although occasionally there may be an absence of either vomiting or purging, it is generally observed that both of these symptoms are present, and in addition acute pain, referable chiefly to the region of the stomach. In the disease referred to, the symptoms commence suddenly in a previously healthy person, and death takes place from strangulation internally. The symptoms are a sudden access of severe pain, chiefly confined to one spot, not in the region of the stomach, as in irritant poisoning, but in the central or lower part of the abdomen; severe and constant vomiting, at first bilious, and afterwards of fecal matter, but in some instances the vomited matter is, throughout, a yellow or green colored liquid. There is obstinate constipation, if we except what may be discharged from the lower bowel. The detection of the disease is commonly not difficult, and a careful inspection of the body will immediately reveal the cause of death. The case in general terminates fatally in three or four days, as there are no means of relieving the strangulation: hence evidence from appearances is rarely absent.

The disease sometimes assumes a chronic form. It commences with colicky pains in the abdomen, and admits of relief by the usual remedies. After a time, purgative medicines cease to act; and the abdomen becomes distended. There is then vomiting, and this speedily assumes a fecal character. These symptoms are unlike those of irritant poisoning, and an analysis of the vomited matters or of the urine, would show the absence of poison. Cases of obstructed (or internally strangulated) intestine, have occasionally given rise to difficult medico-legal inquiries.

Intussusception is a disease which frequently occurs in infants or children. It consists in the reception of one portion of the bowels into another. This leads to a constriction or strangulation of the portion received, and a more or less complete obstruction of the canal. Either nothing is passed per anum, or only a small quantity of blood. It may occur in any part of the bowels; but it is most commonly observed at or near the union of the small with the large intestines. The invaginated portion of bowel varies from an inch to eight or ten inches in length. The disease appears to result from spasm in the intestines, depending on dentition, worms, or other causes of irritation. Purgatives have been known to produce it. The chief symptoms are pain, vomiting, and convulsions: there is no purging. In some cases a lump may be felt in the abdomen in the seat of the disease. Intussusception in an infant has been mistaken for arsenical poisoning, and the mistake nearly led to the conviction of the mother and grandmother of the child, on an unfounded charge of murder. (*Reg.* v. *Dore and Spry,* Central Criminal Court, Aug. 28, 1848; also *Medical Gazette,* Nov. 24, 1848.) In another case of more recent date, which occurred in the family of a medical man, poisoning was alleged, and suspicion fell on some of the servants. A careful inspection of the body of the child showed that there were none of the usual effects of poison, but a portion of the small intestines had become invaginated for several inches. This had led to complete obstruction and the death of the child. A mistaken diagnosis during life may be corrected by a post-mortem examination; but the disease is not always fatal, and in these cases an unjust suspicion may be thrown upon a servant. A correct diagnosis usually presents no difficulty.

An examination of the surface of the abdomen in any of the forms of this disease, may not always suffice to indicate the cause of the sudden illness and death. Nevertheless, the obstinate constipation, with the other symptoms, will in general be sufficient to show that they cannot be ascribed to irritant poison. In these doubtful cases, if the symptoms really be dependent on poison, some connection may be generally established between the last meal taken and the period of their occurrence, and poison will be discovered in the matters vomited.

Colic.—This disease can only be confounded with one variety of irritant poisoning, namely, that induced by the salts of lead. But it is to be observed that the poisonous salts of lead are rarely used criminally, and when they are taken in sufficiently large doses to kill rapidly, the symptoms resembling colic are mixed up with those of irritant poisoning—so as to render it impossible for a practitioner to refer them to the disease alone. It is the *chronic* form of lead-poisoning which resembles colic. This is generally recognizable by the blue line on the gums, the aspect of the patient, and the history of the case.

Hæmatemesis.—In this disease there is neither pain nor purging; and there is a copious discharge of *blood* by vomiting. These characters show that it cannot be easily mistaken for irritant poisoning.

CHAPTER XIV.

DISEASES RESEMBLING NEUROTIC POISONING—CAUSES OF SUDDEN DEATH—APOPLEXY—EPILEPSY—TETANUS FROM DISEASE—FROM STRYCHNIA—FROM LATENT CAUSES—MEANS OF DIAGNOSIS—CASES.

WE have now to consider the diseases which are attended with symptoms resembling those induced by *neurotic* poisons. They are apoplexy, epilepsy, tetanus, diseases of the brain, diseases of the heart, and rupture or distension of the stomach. Indeed, it may be remarked, that every condition of the body in which life is liable to be suddenly destroyed, from whatever cause, may be mistaken for neurotic poisoning. The various causes of *sudden death* should therefore be especially studied by a medical jurist. They are not very numerous, and are principally confined to diseases which affect the brain, heart, and lungs. (For an account of these causes, I must refer the reader to the *Ann. d'Hyg.*, 1838, vol. 2, p. 145; 1843, vol. 2, p. 435; also to the elaborate work of Herrich and Popp, *Der plötzliche Tod aus inneren Ursachen*, Regensburg, 1848.) There is another point to be attended to, namely, those fatal diseases only of these important organs, are likely to be confounded with this form of poisoning, the existence of which had not been previously suspected or announced by the usual attendant symptoms.

Apoplexy.—Those neurotic poisons which act specially on the brain (cerebral poisons), of which we may consider opium to be the type, actually seem to produce this disease. The distinction of apoplexy dependent on disease, from that kind of apoplexy induced by poison, is difficult unless we can obtain a full history of the case. The following circumstances may be remembered in our diagnosis: 1. Apoplexy, as a disease, is sometimes preceded by warning symptoms before the fatal attack comes on. In poisoning, such symptoms would be wanting unless the poison were administered to a person who had already been threatened with apoplexy. 2. Apoplexy, as a disease, does not commonly attack persons under the age of thirty. The fatal cases increase progressively with age, and, according to the researches of Sir G. Burrows, the disease is most common between the ages of sixty and seventy. We shall presently see that there are, however, exceptions to this statement. Poisoning may of course be witnessed in a person of any age. 3. The relation between the time of the attack, and the time at which food or medicine was last taken. Thus if the symptoms of stupor do not come on until five or six hours after some liquid or solid has been swallowed, they are much more likely to be dependent on apoplexy from disease than on poison. This is an important character; but its occurrence is of course purely accidental, for it is by no means unusual that an attack of apoplexy should speedily follow a meal made by a previously healthy person. However, several cases have already been related, which show that this criterion may be sometimes usefully employed to distinguish disease from poisoning (*ante*, p. 83). 4. In apoplexy from disease, it is usually observed that coma (complete insensibility) is at once induced; but in poisoning, this symptom comes on slowly, and is generally preceded by giddiness and stupor. 5. The discovery of poison in the food taken or in the contents of the stomach: this would at once establish the fact of poisoning. 6. The discovery of appearances in the brain indicative of apoplexy, such as effusion of blood or serum. This would negative, *cæteris paribus*, the presumption of poisoning. (See a paper by M. Tardieu, *Ann. d'Hygiène*, 1854, vol. 2, p. 158.)

It is to be observed, that in all cases of disease simulating narcotic (cerebral) poisoning, the disease is assumed to prove fatal—hence there is always an opportunity of searching for the two last-mentioned characters. We do not hear of an attack of apoplexy, from which a person recovers, ever being mistaken for a case of poisoning by opium, but we hear of poisoning by opium being not unfrequently mistaken for apoplexy or convulsions. Dr. Birt Davies has published the two following cases: A person died in what was considered by the physician and surgeon attending, to be a fit; but opium was found in the stomach. A person was attended by a physician and surgeon for some hours. The illness and death were ascribed to and treated by them for apoplexy, but it was proved beyond all doubt that the deceased had died from laudanum. (*Borough Inquests*. Birmingham, 1845.) Such cases are not unfrequent. Deaths have been registered as from "natural causes," when on an examination of the bodies some weeks or years afterwards, the deceased persons have been found to have died from poison. It is impossible to say how many of such cases escape notice for one which is brought to light. These facts show that inquests, in cases of suspicion, without an examination of the body, serve in many instances to conceal rather than to detect crime.

In reference to the age at which apoplexy may make its attack, it may be remarked that healthy girls of the respective ages of sixteen and twenty-two, have died suddenly from this disease. There had been no warning symptoms whatever. I have known a child between two and three years of age die from congestive apoplexy; and the disease has been observed to occur even in infants. Dr. A. Campbell reports a case of apoplexy proving fatal in a child only eleven days old. (*North. Jour. Med.*, Jan. 1845.) Researches on the causes of sudden death in infants have led to the result that apoplexy is more frequent among them than it was formerly supposed to be.

A remarkable case, involving the question—whether death was caused by apoplexy or prussic acid, came before the Senate of Chambery in April, 1843. I allude to that of M. Pralet (*Ann. d'Hyg.*, vol. 26, p. 399; vol. 29, pp. 103, 474), which appears to have excited as much notice on the continent as the case of Sir T. Broughton in England. Several medical witnesses deposed that the deceased had died from prussic acid, administered to him by M. L'Héritier, the accused. Orfila was required to report on the medical evidence. He found that inferences drawn from the application of the analysis of the chemical tests were incorrect, and that the results were essentially negative. Had it not been for his report, it is most probable that the accused would have been convicted, more from the medical *opinions* against him, than from the strength of the medical *facts* of the case. The witnesses appear to have acted on the principle that the whole of their duty consisted in rendering the charge of poisoning probable; whereas, we shall hereafter see that no person can be convicted of this crime on mere *probability*—the fact of poisoning must be made reasonably certain, either by medical or moral evidence, or by both combined.

Epilepsy.—This disease, in some of its symptoms, resembles poisoning by prussic acid only. If the symptoms depend on poison, some liquid or substance must have been taken immediately before their occurrence. If, however, nothing has been taken, the inference would be that the symptoms most probably depended on disease. Death is commonly very rapid in poisoning by prussic acid; but a first attack of epilepsy is not often fatal. If the person has suffered from previous attacks, it is probable, *cæteris paribus*, that the symptoms depend on disease. But epilepsy may by coincidence immediately follow the administration of a draught, or the taking of food—an analysis of the substance taken would then remove any doubt. Supposing none of this to be procurable, then we must remember, that epilepsy simulates narcotic poisoning only when the attack is rapidly fatal. Therefore, an opportunity will always present itself for verifying or rebutting the suspicion of poisoning, by examining the contents of the stomach. I have never met with an instance in which a case of epilepsy was taken for one of narcotic poisoning. The case of Sir T. Broughton (*Reg.* v. *Donellan*, Warwick Assizes, 1781), was considered by some medical men, including John Hunter, who appeared in the defence, to be explicable on a theory of epilepsy; but although no poison was discovered in the body, the facts of the case as well as the conduct of the prisoner were sufficient to prove that poison (laurel-water) was really the cause of death.

Tetanus.—When this disease occurs, it can generally be traced to some cause—a wound, ulcer, burn, or other injury, involving tendinous or nervous structures. Tetanus may arise from causes of a very simple kind, and independently of wounds—as from exposure to wet and cold, or to a current of air. It has been stated that it may even come on without any apparent cause. The cause may, however, have been latent. When it is the result of physical injury it is called *traumatic;* under other circumstances, *idiopathic;* but idiopathic tetanus is by no means common: it manifests itself by trismus (locked jaw), opisthotonos, or emprosthotonos (a tensely curved position of the trunk backwards or forwards, as the result of muscular spasm). The disease sometimes occurs spontaneously in infants, within the first eight or ten days from birth (*trismus nascentium*). Male adults, especially those who are of a robust and vigorous frame, are most liable to attacks of tetanus. According to Dr. Gregory, tetanus from cold occurs, for the most part, within three or four days after exposure to the exciting cause; while traumatic tetanus (from wounds) generally appears about the eighth day. (*Practice of Physic*, 378.) Other observers have found that tetanus from wounds very commonly shows itself from the fourth to the sixth day after the injury; but it may not appear for three or four weeks and then prove fatal. The sooner it commences after an injury, the more rapidly fatal is its course.

Tetanus, or rather tetanic convulsions, may be produced by certain neurotic poisons, which affect the spinal marrow (spinal poisons), especially those belonging to the strychnos tribe—as nux vomica, strychnia, brucia, and all their saline combinations; and there is not only a strong similarity in the symptoms, but an examination of the dead body does not indicate the existence of any well-marked morbid changes in either case. In tetanus from disease or injury, there is a gradual progression of the symptoms. The rigid contraction commences in the muscles of the jaws; it extends to the throat, back of the neck, and, lastly, descends to the abdomen and lower limbs. Professor Colles has remarked that the muscles of the fingers are the last and least affected. (*Lectures on Surgery*, vol. 1, 72.) The rigidity of the muscles continues more or less throughout the disease without intermissions, whereas in tetanus from poisoning there are remissions or intervals of relaxation. A distinction will commonly rest upon the following circumstances: 1. The period of time which has elapsed since any substance, liquid or solid, was swallowed by the patient. 2. The gradual or sudden and violent accession of symptoms—the latter indicating poisoning. In tetanus from disease, the stiffness is first perceived in the jaws; it then progressively extends downwards, attacking the body and limbs, the hands not being commonly affected until the last. In tetanus from poisoning, the attack is preceded by shivering or trembling and gasping for breath, the body and limbs are then simultaneously affected; the hands are clenched, the feet curved, and the jaw is not commonly fixed until a late period, and during a paroxysm. 3. The duration of the case. Tetanus, as a result of local injuries, rarely proves fatal in less than twenty-four hours; and in the idiopathic form, it either does not destroy life, or only after the lapse of many hours or days. In tetanus produced by strychnia given in fatal doses, the person rarely survives two hours after the occurrence of the symptoms. 4. The absence of any wound, ulcer, burn, or personal injury, nervous susceptibility, or exposure to cold, to account for the attack. 5. The discovery of nux vomica, strychnia, brucia, or other poison, in the food, in the matter vomited, or in the contents of the stomach after death.

The case of Miss Abercrombie (1830) is important in reference to the distinction of the symptoms produced by spinal poisons and tetanus as a result of disease. In Miss Abercrombie's case no doubt poisoning with strychnia was mistaken for tetanus from disease. Miss Abercrombie was a healthy young lady in the prime of life; she was induced by her brother-in-law, Wainewright, to insure her life for two years for £3000 in the Imperial Assurance Company, Wainewright having no pecuniary interest in her life. The policy was effected in October, 1830, and she died rather suddenly in the December following. It was not until the lapse of five years that Wainewright brought an action against the company for the amount of the policy (*Wainewright* v. *Bland*, Exchequer, June 29, 1835), and the evidence was such that the jury were equally divided, so that no verdict was given.

The payment of the policy was disputed by the company on the ground of fraud, and the defence was substantially that the lady had died from poison administered by the plaintiff. She had been for a few days indisposed with an hysterical attack, but there was nothing to excite alarm. All that could be learned of her death was that the physician in attendance was suddenly sent for between two and three o'clock. "She was in *convulsions resembling those which were the effect of a wound* (tetanus), and said she was sure she should die, and she went off into convulsions." The physician left the house, returned at four o'clock, and she was then just dead. The appearances presented by the body are imperfectly reported. There was congestion of the vessels of the brain, with some effusion, and the bloodvessels of the stomach were distended. The cause of death was assigned to *convulsions* produced by some oysters which she had eaten for supper, and to wet feet! The Attorney-General put it to the jury whether it was oysters or some poison which had caused this lady's death, a point which they felt unable to decide. (*Medical Gazette*, vol. 16, p. 606.) The cause assigned was quite inadequate to explain this sudden and rapid death.

There can be no doubt that this young woman died from the effects of a dose of strychnia, administered to her shortly before she was seen by her physician, and that he failed to recognize the real cause of the symptoms. The poison was then but little known either in England or France. Tetanus, as it is produced by this poison, is rapidly fatal; but as it arises from wounds or from exposure to cold, it comes on slowly, and is only fatal after some days, and in this case there was no wound or other natural cause to account for its occurrence. Wainewright was subsequently tried on a charge of forgery, convicted and transported for life. He died suddenly of apoplexy in 1852 in Tasmania, while undergoing his sentence as a convict. Before his death, it is reported, he substantially admitted that he had destroyed Miss Abercrombie with strychnia, and had previously killed two other relatives with the same poison—namely, his uncle, Dr. Griffiths, and Mrs. Abercrombie, his wife's mother. Their symptoms were similar, and they all died suddenly. Death was ascribed to heart disease, pressure on the brain, or hysteria!

Tetanus may be the result of *hysteria*, and as such, it is chiefly met with in women, and may be traced to injury to the brain or a peculiar constitution. An attack even in a severe form may be brought on by slight causes producing mental emotion or excitement. It will probably be found on inquiry that the patient has been subject to previous attacks or fits. The spasms of hysteria may be tetanic: but convulsive motions of the limbs more commonly alternate with stiffness or rigidity, and the attack is generally attended with loss of consciousness. In poisoning, the patient retains consciousness, and the paroxysms, if frequent and severe, are generally fatal: in cases of hysteria or hysterical excitement, the attack is not fatal, but the patient speedily recovers. Such, at least, is the result of experience up to the present time.

The case of J. P. Cook (*Reg.* v. *W. Palmer*, Central Criminal Court, May, 1856), falls under the distinctive criteria above pointed out. There was no wound or personal injury. There was no reason to suppose that the tetanic convulsions from which the deceased had suffered, were of the idiopathic kind, *i. e.*, that they had arisen from exposure to wet or cold,—or from excitement as a result of his having won a race a week before the attack. Deceased had had some pills administered to him by the prisoner, at a time which would correspond to the interval that precedes the action of strychnia. The prisoner had secretly purchased strychnia on the morning of the day on which deceased died, and could not account for the purchase of it or state what he had done with it! The symptoms were sudden and violent, developed over the entire body and limbs in a few minutes, and they proved fatal in *twenty minutes!* The pills could not be obtained for analysis, and no strychnia was found in the stomach, which had been cut from end to end, and the fluid contents lost, by the deliberate act of the prisoner, during the post-mortem examination. The physiological and pathological evidence, however, that deceased had died from strychnia was considered to be conclusive, and on this evidence the prisoner was convicted and executed. (See *Guy's Hospital Reports*, October, 1856, *On Poisoning by Strychnia.*)

The great point of contention in this case was: Did the tetanic symptoms under which the deceased had died, depend on disease or poison? Brodie, Christison, and Todd, and other eminent authorities, agreed that, when taken as a whole, they were not in accordance with any known form of disease, but were in perfect accordance with the effects of strychnia. The opinions given by some witnesses in the defence were to the effect that the symptoms were consistent with strychnia-poisoning; and although some professed to perceive differences, the records of medical experience proved that these differences had no real existence. Barristers may well mistrust the evidence of experts, when, in a case so clear as this, one physician could be found to swear that the symptoms under which the man died were those of angina pectoris, because the heart was empty; and another physician, also employed for the defence, assigned death to "epilepsy with tetanic complications." If these gentlemen had been called in to attend on this victim of secret poisoning while living, it is quite obvious that they would have had no suspicion of poisoning. One would have certified the cause of death as angina pectoris, and the other as epilepsy! If physicians of some standing and profound experts can thus overlook an ordinary case of poisoning with strychnia, it is not surprising that general medical practitioners, who have not given special attention to the subject of toxicology, should fall into the error of granting erroneous medical certificates, and of certifying that death from arsenic or opium was due to cholera, convulsions, or apoplexy.

Whenever symptoms resembling tetanus appear suddenly with severity and prove fatal, a minute investigation should be made into the history of the patient and all the circumstances attending the attack. In traumatic tetanus the cause is often overlooked: a small splinter of wood or metal may penetrate the palm of the hand or sole of the foot, the wound may completely heal over the foreign body, and no suspicion of latent mischief may exist. In the spring of 1858, a man was admitted into Guy's Hospital suffering under tetanus; he died in three days with the usual symptoms. The cause of the tetanus was traced to a wound in the hand received a *month* before; this was perfectly healed, and from the length of time which had elapsed it was not thought of importance, but on cutting into the wounded part after death, a piece of rusty iron was found imbedded therein, and pressing on a nerve. In another case a young man was admitted, and died from tetanus in ten hours. On inspection, it was found that a slight wound in the hand, received shortly before, had completely healed and inclosed a splinter of wood—the exciting cause of the disease. The facts connected with these cases were not consistent with the theory of poisoning by strychnia, nevertheless, they show that after the complete healing of a wound, and at a long interval, traumatic tetanus in a fatal form may insidiously make its appearance. Two similar cases have been communicated to me by Dr. G. Johnson. (*Brit. Med. Jour.*, Nov. 1872, p. 594.) For other cases showing how easily the true cause of the symptoms may be overlooked, see *Principles and Practice of Medical Jurisprudence*, 2d ed., vol. 1, p. 579.

On the other hand, poison may have been unconsciously taken, and the symptoms referred to disease. This occurred in the case of Assistant-Surgeon Bond, at Moulmein, in March, 1858. This gentleman, intending to take two aperient pills, swallowed by mistake two pills containing in each one grain of strychnia. He was seized with violent tetanic convulsions and opisthotonos, and died in less than *two hours* after the commencement of the symptoms. Deceased was unconscious of having made a mistake in taking his medicine, even to the last, and his medical friends had at the time no suspicion that strychnia was the cause of the symptoms. His illness was attributed at first to irritation of the spinal cord proceeding to inflammation, arising from his having been exposed to a current of cold air while in a heated state. It was not until after he had expired that it was found he had taken pills containing strychnia in place of aperient pills. I am indebted to his medical attendant for this information. But for the accidental discovery of the strychnia pills, the tetanus might have been referred to hysterical excitement, or some "undiscoverable disease" of the spinal marrow, although its characters were clearly those of strychnia-poisoning.

A case reported by Dr. Lonsdale (*Edinburgh Monthly Journal of Medicine*, Feb. 1855, p. 117) shows the dangerous facility with which tetanus, as a result of poisoning with strychnia, may be overlooked. In November, 1854, a man, æt. fifty-nine, went, apparently in his usual health, early in the morning to bathe in the river Esk, near Carlisle. About eight o'clock he was seen walking home, and on reaching his own house, he complained of severe illness, was violently cramped, and declared himself dying. A doctor was immediately called, who prescribed a pill, but the man, after exhibiting some marked symptoms, died within thirty or forty minutes of his arrival at home. As deceased had often suffered from inward complaints, was a frequent patient at a county charity, and had that morning (in November) taken an early bath in the river, it was considered by his neighbors that the cold had struck him, and his disease was looked upon as sudden death from natural causes! An inquiry before the coroner, however, led to the discovery that the man had been accidentally poisoned with strychnia. The physician who saw deceased during his illness stated that when called to him at half-past eight A.M., he was laboring under violent spasms, which almost entirely subsided in from four to seven or eight minutes. During the spasms the body was extended, with the limbs separated, stiff, and rigid, and there was a violent shaking of the whole body. At first the spasms were most marked down the back and legs, but in the course of from ten to fifteen minutes they fixed upon the chest, and violent tetanus supervened with fixation of the muscles of respiration, and in this state the patient died. Deceased was anxious, agitated, and felt certain of impending death. His intellect was perfectly clear. The eyes protruded, the pupils were dilated, and the mouth was spasmodically closed. The face and hands were livid, and the surface below the natural temperature. It turned out, on due inquiry, that, on his return from the river, deceased had called at a druggist's for a strong dose of purgative medicine, and the druggist, as it was afterwards shown, had served him with a grain and a half of strychnia by mistake for jalapine! The symptoms and death had been caused by poison, not by natural disease. (See *Lancet*, March 27th, 1858, p. 318.)

These cases show the very difficult position in which a medical jurist may be placed. On the one hand, he may assign to poison symptoms which are really caused by disease; on the other, he may be induced, from an imperfect knowledge of the facts, to refer to disease, a death which is actually caused by poison. In the latter case, he not only lends his science to conceal a murder, but he advertises a method by which a number of lives may be easily sacrificed, and criminals escape with impunity! Each case must be decided by *all* the circumstances, medical and moral, which attend it. An implicit faith in a few symptoms will expose a man to the risk of setting free a murderer, or of leading to the conviction of an innocent person. It is proper to remember on such occasions, that a charge of murder by poison is not likely to be raised unless there are "suspicious circumstances," or unless death has taken place suddenly with violent symptoms, in the entire absence of any apparent or probable natural cause. In the cases of Assistant-Surgeon Bond, and of the man whose death is reported by Dr. Lonsdale, there was *primâ facie* evidence for believing that these persons had died of poison. In other cases of strychnia-poisoning, there has been an absence of such evidence. As cholera may sometimes put on the features of poisoning by arsenic, so may tetanus occasionally put on the features of poisoning by strychnia. In either instance a close sifting of all the facts is necessary before we can form a correct medical opinion; the exceptional resemblance which we occasionally meet with furnishes no reason for abandoning every future case as unsolvable by medical science, and thus giving free scope to secret murder in its worst and most dangerous form.

The remarks above made, have been chiefly restricted to poisoning with strychnia; but tetanus or tetanic convulsions may be an effect of other poisons. Arsenic, tartarized antimony, and prussic acid have been known to produce them. In these cases, however, the tetanic are either preceded or followed by other symptoms of a special kind, which rėmove any difficulty in the formation of an opinion. The presence of poison in the vomited matters during life, or in the contents of the viscera after death, will also aid a medical practitioner in arriving at a just conclusion.

CHAPTER XV.

DISEASES RESEMBLING NEUROTIC POISONING—CONVULSIONS IN CHILDREN—DISEASES OF THE BRAIN AND SPINAL MARROW—CEREBRO-SPINAL MENINGITIS—DISEASES OF THE HEART—SUDDEN DEATH FROM SYNCOPAL ASPHYXIA—DISTENSION AND RUPTURE OF THE STOMACH—RUPTURE OF THE GALL-BLADDER—EMBOLISM.

Convulsions.—This is a frequent cause of death among children. Convulsions (or fits, as they are vulgarly termed) may arise from the action of poisons, especially of those belonging to the neurotic class, or from the effects of disease. As they sometimes attack children suddenly, and prove fatal rapidly, a suspicion may arise that death has been caused by poison administered to the child. Many cases of this kind have been referred to me for investigation; and, from the frequency of their occurrence, and the unjust suspicions to which they may give rise, it is the duty of a practitioner to make himself well acquainted with the ordinary causes of convulsions. Medical writers have divided them into symptomatic and idiopathic. They are most commonly symptomatic, *i. e.*, depending on some disease or morbid condition of the system, such as dentition, repelled eruptions of the skin, water on the brain, exposure to cold, indigestion, worms, accumulation of fæces, improper food, or overdistension of the stomach and bowels with food; and even a peculiar condition of the nurse's milk may become a cause. The younger and more irritable the child, the greater is its liability to an attack; and in such cases, the slightest cause of irritation to the nervous system may lead to it. Children are considered to be most liable to the disease during the appearance of the first set of teeth, *i. e.*, from the fifth to the eighteenth month.

When the convulsions cannot be traced to any of the causes above assigned, they are described as idiopathic, and are commonly referred to some primary disease of the brain; and this organ, after death, may be found in a state of congestion. Idiopathic convulsions sometimes run through their course and cause death very rapidly; but it is not at all improbable that, by diligent inquiry, some cause may be generally found. Dr. Underwood met with several instances in which fine healthy children died suddenly from convulsions soon after they had been overfed by their nurses. This is, no doubt, a common cause of death in infants.

Convulsions in children are a very common effect of the action of overdoses of opium; and they are not easily distinguished from those which arise from natural causes. During the fit the eyes are distorted, and the pupils contracted or dilated. The spasm may affect the breathing; the jaws are closed, and saliva, in a frothy state, escapes from the mouth. There may be also stertorous or snoring breathing; and, from impeded respiration, the tongue, face, and the surface of the skin become livid, owing to imperfect aeration of the blood, and the child may die asphyxiated. Under prompt and appropriate treatment, except when it depends on poison unsuspected, the attack may be alleviated, and the child recover. When a neurotic poison is the cause, it will be found that some substance, either liquid or solid, has been given to the child not long before. When laudanum has been given, it may be perceived by the smell. Except by a chemical analysis of the food and the contents of the stomach, it is by no means easy to distinguish disease from poisoning. A contracted state of the pupils will indicate, *cæteris paribus*, that a preparation of opium is the cause.

In a case which occurred in December, 1846, a suspicion arose that two children had been poisoned, from the singular fact that they had died within a short period of each other, having been previously well. One, an infant, aged seven months, was found by the mother at 6 A.M. in a fit. It was livid in the face, frothing at the mouth, and its limbs were drawn up and rigid. She immediately took it to a person living in the same house, but it remained insensible until it died about two hours afterwards. The child appeared well when put to bed on the previous night, and had had its last meal (boiled bread and milk) about 7 P.M. The chief appearances on inspection, were congestion of the brain and lungs; there was slight redness of the stomach. On the same morning and at about the same time, the other child, aged fifteen months, was found by the mother insensible, dark in the face, and struggling for breath. The child died five minutes after it was found in this state. On inspection, the only appearance was general congestion of the brain. The stomachs and their contents, as well as a portion of the food given to the children the night before, were examined for opium as well as other poisons which were likely to have occasioned the symptoms; but no trace of poison could be found. There was no moral evidence to show that poison had been given; none was detected in the food; and had it been given by the mother, who found the children dying early in the morning, it is probable that, as there had been no vomiting, and death was rapid, the poison would have been discovered by the odor, either in the stomach or its contents, or by the usual tests. The opinion which I gave was, that death had resulted from convulsions, probably produced by a congested state of the brain. The most remarkable feature in this case was the coincidence in seizure and the time of death; and, but for the good character of the parents and the results of a chemical examination of the food and the viscera, it would have been difficult to have satisfied the neighbors that the children had not been destroyed by poison. The jury returned a verdict of death from natural causes.

It is not sufficient on these occasions to assign death to *convulsions;* the cause should if possible be indicated. The convulsions may really have arisen from some poison administered to a child: for whatever affects strongly the nervous system of a child, may bring on an attack of convulsions which may prove fatal. In *Reg.* v. *Connell* (Cent. Crim. Court, Nov. 1852) the prisoner, a female servant, gave a quantity of sulphuric acid to an infant. Owing to the local effect of the acid the child was not able to take food; it became very weak, and died on the sixteenth day, from convulsions. The medical witness assigned this fatal attack to the poison, because the child had not been before subject to fits, and the cause appeared adequate. It was admitted that convulsions might arise from other and natural causes, and on the doubt thus raised the prisoner was acquitted.

Diseases of the Brain and Spinal Marrow.—Among these diseases may be mentioned inflammation of the brain and its membranes, hypertrophy, and the formation of tumors. Such diseases are of a very insidious nature—they sometimes give no warning of their presence, until the person, who may be in his usual health, is suddenly seized with stupor, followed by coma, or paralysis, and rapidly dies. All such cases resemble poisoning with morphia or opium: they can be distinguished only by the discovery of the affirmative characters of disease, on an examination of the body, and an absence of poison from the stomach. But the time at which the symptoms appear after a meal, and the rapidity of death, will in many instances allow a practitioner to form a satisfactory distinction. This subject has been elsewhere considered (*ante*, p. 82).

Cerebro-spinal Meningitis.—There is an affection of the brain and spinal marrow known under the above name, the symptoms of which it is supposed might be mistaken for poisoning with strychnia. In a remarkable case tried at Annapolis, Md., Dec. 1871 (trial of Mrs. E. Wharton for the murder by poison of General Ketchum), this disease was brought forward on the part of the defence to account for the symptoms and death of the deceased, while it was alleged for the prosecution that he had died from the effects of poisonous doses of tartar emetic administered to him by the prisoner.

Cerebro-spinal meningitis consists essentially of inflammation of the membranes of the brain and spinal marrow. Dr. G. B. Wood, of Philadelphia, states that in severe cases (those which are likely to be confounded with poisoning) the attack is ushered in with a chill, during which the patient complains of acute abdominal pains, is not unfrequently affected with vomiting and purging, and sometimes sinks into a state resembling the collapse of cholera. In milder cases the earlier symptoms are a sense of fatigue; headache; pain in the neck, back, along the whole of the spine; stiffness of the jaws, with some difficulty of swallowing.

As the disease progresses the headache becomes more violent; there is great sensitiveness to light and sound, with delirium and convulsions. These are the cerebral symptoms, while the spinal symptoms are manifested by rigid spasms or cramps—the head being drawn stiffly backwards and the whole body sometimes becoming as rigid as a board (opisthotonos). The countenance has in some cases presented a tetanic expression or grin (*risus sardonicus*), also observed in strychnia-poisoning. There is great febrile excitement, and a hot skin, frequent pulse, great thirst, with vomiting and tenderness in the region of the stomach. Death may take place in severe cases in from two to five days. The chief post-mortem appearances are inflammation and thickening of the membranes of the brain and spinal cord, with effusion of turbid serum or lymph. The pia mater is reddened, injected, or swollen. In some instances there has been an absence of any cerebral lesion: no induration, softening or unusual redness was observed. (Wood's *Treatise of the Practice of Medicine*, vol. 2, p. 759.)

We may first differentiate these symptoms from those of strychnia. Some article of food or medicine containing the poison must have been taken shortly before the attack. The symptoms in strychnia-poisoning commence with tremors and shivering—tetanic convulsions take place suddenly throughout the whole of the body, succeeding each other rapidly at intervals—the intellect preserved—death within an hour or two after their commencement—no vomiting or purging, no febrile symptoms. The differences are here sufficiently marked without reference to the aid of chemistry. By the detection of strychnia in the food or in the body, the real nature of the case would be at once evident.

On the trial of Mrs. E. Wharton, at Annapolis, Md. (*ante*, p. 110), it was alleged that cerebro-spinal meningitis had been mistaken for poisoning with tartar emetic. The deceased, General Ketchum, died after a few days' illness, and the prisoner was charged with having administered to him during his illness one or more doses of tartar emetic, thereby causing his death. The trial lasted fifty-two days, and owing to the social position of the parties, and the number of scientific witnesses called for the prosecution and defence, it excited an unusual degree of interest.

I am indebted to Dr. Chew, a witness for the prosecution, for a very concise and clear account of this case. On the evening of Saturday, June 24, 1871, the General, who was in his usual health, came on a visit to the accused, Mrs. Wharton. He had supper at 9 o'clock with the family, went to bed at 11, was attacked in the night apparently with cholera, and was obliged to leave his room. On Sunday morning, June 25, he got up, went out, but soon returned complaining of feeling unwell. He remained in his room until 8 or 9 o'clock, when he took a glass of lemonade with brandy in it. During the night symptoms of cholera again obliged him to leave his room. On Monday, the 26th, he suffered from nausea, which increased to such a degree that at 4 P.M. Dr. Williams was summoned to him and found him much prostrated, with a cool clammy skin, rapid and feeble pulse, and vomiting repeatedly. He was relieved by medicines. On Tuesday, the 27th, at 10 A.M., he was again seen by Dr. Williams, to whom he stated he was well enough to return to Washington, and would go that day. He did not go, but on the evening of this day he was heard to vomit violently shortly after taking some porter. On Wednesday, the 28th, at 10 A.M., he was seen by Dr. Williams, and found in a semi-comatose state, imperfectly conscious; with a clammy skin and a feeble pulse; face livid; pupils natural but insensible to light, and the muscles of the neck, back, and lower extremities were rigid. When asked in a loud tone how he felt, he replied "tolerably," and relapsed into a drowsy state. The urine was tested and found free from albumen. At 1 o'clock P.M. two tablespoonfuls of liquid were given to him, and in fifteen minutes tetanic convulsions ensued. The jaws were firmly clenched, and the convulsions increased in violence until his death at 3 o'clock P.M.

On a post-mortem examination patches of redness, indicative of irritation, were found in various parts of the intestines. The lungs, heart, liver, kidneys, and spleen were healthy. There was some passive congestion of the pia mater, but there was no effusion of fluid or lymph in the ventricles or upon the membranes of the brain. The upper part of the spinal cord was examined to the extent of two inches, and found free from disease except slight congestion. The stomach contained nearly four ounces of a turbid brownish fluid; the interior presented nothing very marked.

The contents of the stomach were examined for strychnia, arsenic, and antimony. The two first were not present, but antimony is stated to have been found, and produced as a brownish-red sulphide, possessing the properties usually assigned to this compound, with the exception that no metallic antimony was obtained from it or from the liquid which yielded it, although it was calculated that the fluid of the stomach contained twenty grains of tartar emetic. Some sediment in a glass, it is also stated, contained tartar emetic.

It was proved that the accused had recently purchased tartar emetic, and that she had had it in her possession at the time of deceased's visit. It was contended that she had secretly administered it to the deceased during his illness; that she had a motive for the act, and that he had really died from the effects of this poison. The jury were not satisfied with the evidence, and the prisoner was acquitted.

Several of the medical witnesses for the defence, while agreeing that poison was not the cause of death, adopted the view that the symptoms, taken as a whole, were consistent with an attack of cerebro-spinal meningitis—others took the more cautious view, that death was consistent with natural causes, without defining the specific cause, and that it was not owing to poison.

The medico-legal questions which present themselves in this case are: 1. Were the symptoms such as we are accustomed to see in cases of acute poisoning by tartar emetic? 2. Did the chemical evidence conclusively prove the presence of antimony in the body? This would be the marked point of distinction between poisoning and disease.

During the first three days of his illness, the symptoms suffered by deceased were such as might have arisen from a severe attack of cholera. On the fourth and last day some liquid was given to him, and in fifteen minutes afterwards tetanic convulsions with trismus ensued, and he died in convulsions in two hours. In an acute case of poisoning with tartar emetic, there is a strong metallic taste; heat and constriction, with a burning pain in the throat; pain in the stomach; incessant vomiting and profuse purging of a bilious character, with the usual signs of depression and collapse. Spasms and convulsions have been observed, but these symptoms have been rather of an exceptional kind, and have usually been preceded by the violent irritant action of the poison on the stomach and bowels. Taken as a whole, the symptoms in General Ketchum's case were not consistent with acute poisoning with tartar emetic.

A skilled physician, Dr. Williams, attended the deceased from the Monday until the Wednesday, and treated the case as one of disease, and not of antimonial poisoning. About an hour before the patient's death, he expressed a suspicion that the General had been poisoned. Five hours before death the patient's urine had been tested—not for metallic poison, but for albumen—a grave omission, considering the subsequent proceedings in the case. The matters vomited during the illness were not examined for antimony or any other poison, a fact which can only be explained by the non-existence at that time of any suspicion that poison was the cause of the symptoms.

Of the appearances in the body, nothing need be said. They proved nothing for or against the theory of poisoning.

The second question is purely chemical, and the reply to it may be summed up in a very few words: When a case is left at all doubtful, from symptoms and appearances, the chemical evidence should be unusually clear, complete, and conclusive, in order to justify a conviction. It should not fall short of the most complete demonstration of the presence of the alleged poison. It was the more necessary in this case, because neither the vomited liquids nor the urine had been tested for poison.

Although twenty grains of tartar emetic were estimated to be present in the four ounces of fluid contents of the stomach, the analysis for antimony was carried only to the extent of producing a brown precipitate with sulphuretted hydrogen. This was inferred to be sulphide of antimony by its solubility in hydrochloric acid and subsequent precipitation by water. No metal was produced from it or from the original contents of the stomach, although a single grain would have been ample to furnish the most complete evidence of the presence of antimony.

Another part of the chemical evidence is inconsistent with the statement that the precipitate obtained was sulphide of antimony. The witness admitted that he had examined the contents of the stomach for arsenic by Reinsch's process (metallic copper and hydrochloric acid) but it "gave no result." Had antimony been really present even in a small fraction of a grain, it would have been visibly deposited on the copper!

The liver and other organs were not examined for absorbed antimony until after the trial had commenced, and therefore too late to be made available as legal evidence.

There may have been strong moral or political reasons for imputing poisoning in this case, but with the medical facts as reported, death from poison was not conclusively proved, either by the pathological or chemical evidence. The jury were, therefore, justified in acquitting the prisoner of the charge. (*Report of Trial of Mrs. E. G. Wharton, on the charge of poisoning General W. S. Ketchum, Dec.* 1871. Jan. 1872. Medical survey of the case by Dr. S. C. Chew; also a report by Dr. Reese, *Amer. Jour. Med. Sci.*, April, 1872, p. 329.)

Diseases of the Heart.—The heart is subject to many diseases, which present the same insidious characters as those of the brain. Thus they may remain for a long time latent, and then suddenly destroy life. They are only likely to be confounded with poisoning by prussic acid, owing to the rapidity with which death takes place. In all these cases, therefore, if the fatal attack occur suddenly some hours after food or medicine has been taken, there can be no reason for attributing it to poison. It is only when by a coincidence, the symptoms appear immediately after something has been swallowed by the patient, that any doubt of the cause to which they may be due, can arise; and here, the doubt would be speedily removed by an examination of the body. We must not expect, however, that in these fatal affections of the heart, well-marked appearances will always be found. Some pathologists have described a singular condition of this organ, under which a person is liable to die suddenly after experiencing nausea and giddiness. In such cases, the muscular substance of the heart has been found only preternaturally flaccid, and its cavities empty. This has been called by Mr. Chevalier *Idiopathic asphyxia*, and others have termed it *Syncopal asphyxia*. It does not appear to be very common, for very little is known concerning it, or on what the cause of death really depends. In regard to its recognition in suspected cases, all we can say is, that if poisoning be not clearly negatived by concurrent circumstances, its usual affirmative characters are entirely wanting. The emptiness of the heart appears to be the chief indication of this variety of sudden death. An emptiness of this organ, however, is met with in other cases. It has been frequently observed in death from strychnia: and has here been supposed to depend on spasm. It is probable, however, that in all these cases there is a slight action of the heart in the act of dying, by which its cavities are emptied, after the vessels of the lungs have ceased to transmit blood.

In the case of Lord George Bentinck (Sept. 1848), emptiness of the heart was the only remarkable appearance found in the body. The deceased had died suddenly while taking a walk. He had not partaken of food for many hours. He was found dead lying flat on his face, and both of his hands were under him; his stick was firmly grasped in one hand. The body had been lying exposed five or six hours, and there was only the usual rigidity of death. At first it was suspected he had died from poison, but there was nothing to support this view. It was probably a sudden attack of syncopal asphyxia which had caused his death while in the act of walking.

The possession or purchase of poison may be a mere coincidence with sudden death from natural causes. Mr. Stedman, of Guildford, was called to see a woman, æt. 30, who was found dead in her bed. There was nothing externally to indicate a violent death, and the only appearances in the body were congestion of the lungs with an enlarged pale and flabby heart. On the day of her death, she had secretly procured a packet of Battle's vermin killer (strychnia). She was seen in her bedroom in her usual health, and half an hour after she had received the poison, she was found dead.

No trace of the poison could be found, nor any cup or vessel out of which it could have been taken. The contents of the stomach were examined by Dr. Bernays. There was no strychnia or other poison present, and none of the blue coloring matter with which the strychnia sold was mixed. (*Med. Times and Gaz.*, 1865, Jan. 13, p. 34.) The cause of death was properly assigned by Mr. Stedman to failure of the heart's action from disease and congestion of the lungs. A case like this should convey a caution to those who allow their medical opinions to be influenced by the proof of purchase or possession of poison.

Distension of the Stomach.—This is by no means an unfrequent cause of sudden death: it may occur in infancy or at any age. In some instances, the distension of this organ appears to act by inducing apoplexy, the usual marks of that disease being found in the brain. In other cases, death appears to be due to a fatal impression analogous to shock, arising simply from the excessive mechanical distension of the organ: it is not surprising that a suspicion of poisoning should occasionally arise under such circumstance. I have known several instances which have occurred in this metropolis, where persons went to bed in their usual health after eating a full supper and were found dead on the following morning. On dissection, no marked changes were discovered, excepting, in some cases, slight congestion of the cerebral vessels. The most striking appearance was the enormously distended state of the stomach itself. In December, 1839, a woman, aged 22, after eating a hearty supper, retired to rest. In about two hours she was found insensible and died in the course of a few minutes. There was no examination of the body; although it is difficult to understand why, without it, there should have been any inquest—as the cause of death, which was probably due to the distension of the stomach, was left unexplained. In April, 1841, a man, aged 34, ate a full breakfast, consisting of three-quarters of a pound of beef with bread, and a pint and a half of coffee. In a few minutes afterwards, he sat on a barrel to rest himself, but almost immediately fell backwards and expired. This cause of death may be met with in persons of all ages. In November, 1842, a girl, aged 13, ate a full breakfast; and about an hour afterwards she became insensible, and died in the course of a short time. The only cause which could be assigned for her death was overdistension of the stomach with food, probably leading to apoplexy.

A case of a somewhat similar kind was the subject of an inquest at Hoxton, in April, 1865. A man, æt. 75, according to the evidence, ate, on a Good Friday, fourteen buns. He complained of pain in the stomach, and some hours afterwards he was found dead in bed. On examination, there was a greatly distended stomach, with congestion of the brain and disease of the heart.

Another cause of sudden death in these cases may be asphyxia from the vomiting of a portion of the contents of the stomach into the back of the throat, and a want of power to expel them. This is chiefly observed in persons who go to bed in a state of drunkenness. Portions of the vomited food are thus drawn by aspiration into the air-passages and suffocation ensues. In one instance, a drunken man was thus suffocated by a thin piece of potato skin being drawn over and closing the glottis. In another case, communicated to me by Dr. R. Elliott (May, 1874), a lady, who had been rendered unconscious by chloroform, died from suffocation, as a result of the food finding its way into the air-passages. She had vomited, but owing to her state of insensibility, had not had power to expel the vomited matter from the fauces. Cases of this kind are readily detected by a careful inspection of the body.

Rupture of the Stomach has been observed to occur sometimes as a consequence of overdistension, combined with efforts at vomiting; although in other instances the rupture has taken place from disease, when there was but little food found in the stomach. Death is, of course, a speedy consequence of this accident: hence no difficulty can arise in practice with regard to it, because an examination of the body would enable a practitioner at once to determine the cause. (For a fatal case of this kind, in which there was no apparent disease of the organ, see *Medical Gazette*, vol. 2, p. 182.)

Rupture of the Gall-bladder and gall-ducts, as well as of the impregnated *uterus*, or its appendages, may also suddenly give rise to alarming symptoms of a suspicious kind in a previously healthy person. Death in such cases commonly takes place from peritonitis. The rules for forming a diagnosis are similar to those already described: an examination of the body will show the cause of death, as in the following case, communicated to me, in May, 1870, by a former pupil, then practicing in Rome:

On May 9, 1870, a young lady, residing in Rome, and supposed to be some months advanced in pregnancy, died very suddenly soon after taking some medicine prescribed for her by a physician. She had enjoyed excellent health, with the exception of being occasionally subject to slight abdominal pains threatening abortion, and to relieve these pains a physician was consulted. It seems that she had aborted on a previous occasion. She was found to be in a state of great depression, but not suffering at the time from any dangerous symptoms. The physician had prescribed a sedative medicine, of which the patient had taken only three doses in teaspoonfuls when she fell into a deep sleep, and in this state she died, the symptoms of depression not having been relieved. The family attributed her death to some mistake made by the druggist who prepared the medicine. The tribunal before which the charge was laid, directed an inspection of the body. The result was, that a quantity of blood was found effused in the lower part of the abdomen. This had obviously arisen from the rupture of a tumor, containing an embryo, of which the remains were found in the pelvis, in the midst of the clots of blood. It appeared to be of only a few weeks' development. The body had been contained in a cyst external to the uterus, which had suddenly given way and had thus led to fatal hemorrhage. It was the suddenness of death soon after taking medicine, without any preceding symptoms of illness or any other obvious cause, except the medicine, to account for her condition, that gave rise to the inquiry. A post-mortem examination revealed, as it always will under these circumstances, the real cause of death.

Embolism.—This is a cause of sudden death in diseases of exhaustion and debility, in croup, and also in the pregnant or puerperal states. It consists in the obstruction of the circulation, by the formation of a clot or plug of fibrin in the larger or smaller vessels, or in the heart itself (ἔμβολος, a plug). The fibrinous substance which thus mechanically impedes circulation, may be formed suddenly or gradually, and the symptoms vary accordingly. In the former case, the patient may die in the act of sitting up in bed or in making a slight exertion. The breathing is hurried and gasping, the skin pale and cold, the pulse frequent, small, and intermittent, and there are all the signs of extreme collapse. Some of those suddenly fatal cases which have been attributed to syncope or asphyxia have been really due to embolism. Poisoning might be supposed to be the cause of death when anything had been administered to the deceased shortly before the attack, or when moral circumstances justified a suspicion of foul play. As the death is assumed to be sudden, any poison, if taken, would necessarily be detected. In the absence of this, and in the discovery of a fibrinous plug in the heart or great vessels, we should have clear evidence of the cause of death. Dr. R. Elliott, coroner for Carlisle, communicated to me a case which was the subject of an inquest in 1874, in which death was due to embolism, although in the first instance there was reason to suspect the administration of poison.

When called to examine a case of suspected narcotic poisoning, and the symptoms have occurred soon after a meal, a practitioner must remember that, although a full meal is a very common exciting cause of apoplexy, this is not the case with any simple medicine, liquid or solid, which may have been swallowed by the patient. Should the symptoms follow the taking of a draught, pill, or any kind of medicine, the circumstances become much more suspicious, because the occurrence of apoplexy in such a case would be a pure coincidence; all we can say is, that it may happen—in proof of which we may refer to a case mentioned (*ante*, page 87), and then we require other circumstances to aid our judgment. In such cases it can never be assumed that the medicine taken was the cause of the symptoms, unless we suppose it to have been a poison; while, when the symptoms follow an ordinary meal, apoplexy may be a natural result; at least it is not absolutely necessary, in order to account for them, to suppose that the food actually contained any poison.

CHAPTER XVI.

EVIDENCE OF POISONING IN THE DEAD BODY—TIME WITHIN WHICH POISONS PROVE FATAL—CHRONIC POISONING—DIFFICULTY OF DIAGNOSIS—ACCUMULATIVE POISONS—APPEARANCES IN THE DEAD BODY—EXTERNAL AND INTERNAL—REDNESS OF THE MUCOUS MEMBRANE—ULCERATION—SOFTENING.

SUPPOSING that the person is *dead*, and we are required to determine whether the case is one of poisoning or not, we must, in the first instance, endeavor to ascertain all the particulars which have been described as indicative of poisoning noticed in the *living* subject (p. 93). Should the deceased have died from poison, the circumstances of the attack and the symptoms preceding death, ought to correspond with the usual characters of poisoning; and in these investigations it is well to bear in mind the following rule: There is no one symptom or pathological condition which is peculiar to poisoning; but at the same time there is no disease which presents *all* those characters that are met with in a special case of poisoning. The points which should be specially noticed under these circumstances in the *living* body are described at p. 93, *ante*. The additional evidence to be derived from the *death* of the person may be considered under the following heads:

1. *The Time at which Death takes place after the First Occurrence of Symptoms.*—This question requires examination, because the more common poisons, when taken in fatal doses, generally produce their fatal effects within certain periods of time. By an attention to this point, we may, in some instances, be enabled to negative a charge of poisoning, and in others to form an opinion of the kind of poison which has been taken. In a court of law, a medical practitioner is often required to state the usual *period of time* within which a poison proves fatal. It is to be observed that not only do poisons differ from each other in this respect, but the same substance, according to the form or quantity in which it has been taken, will differ in the rapidity of its action. A large dose of strong prussic acid, *i. e.*, from half an ounce to an ounce, may destroy life in less than two minutes. In ordinary cases of poisoning by this substance, a person dies, *i. e.*, all signs of life have commonly ceased, in from ten to twenty minutes—if he survive half an hour, there is some hope of recovery. In the cases of the seven Parisian epileptics, accidentally poisoned by a similar dose of prussic acid prescribed medicinally, the first died in about twenty minutes, the seventh survived three-quarters of an hour. (See PRUSSIC ACID, *post.*) Oxalic acid, one of the most energetic of the common poisons, when taken in a dose of from half an ounce to an ounce, may destroy life in from ten minutes to an hour; if the poison be not perfectly dissolved when swallowed, it is a longer time in proving fatal. The strong mineral acids, in poisonous doses, destroy life in about eighteen or twenty-four hours. Arsenic, under the form of arsenious acid (white arsenic),

operates fatally in from eighteen hours to three or four days. It has, however, in more than one instance, killed a person in two hours; although this is by no means common. Opium, either as a solid or under the form of laudanum, commonly proves fatal in from six to twelve hours; but it has been known, in several instances, to destroy life in less than three hours; those who survive the effects of this poison for twelve hours are considered to have a fair chance of recovery. Strychnia has proved fatal in from twenty minutes to six hours after the poison has been taken. This must be understood to be merely a statement of the average results, as nearly, perhaps, as we are warranted in giving an opinion; but a medical jurist will of course be aware that the fatal period may be protracted or shortened, according to all those circumstances which have been elsewhere stated to affect the action of poisons (*ante*, p. 78).

There are various forms which this question may assume in a court of law; the death of a person, alleged to have taken poison, may have occurred too rapidly or too slowly to justify a suspicion of poisoning. The following case may serve as an illustration: A woman of the name of Russell was tried and convicted at the Lewes Summer Assizes, in 1826, for the murder of her husband, by poisoning him with arsenic. The poison was detected in the stomach; but the fact of poisoning was disputed by some medical witnesses, for this among other reasons,—that the deceased had died *three* hours after the only meal at which the poison could have been administered to him. The authority of Sir A. Cooper and others was cited to show that, according to their experience, they had never known a case to prove fatal in less than seven hours. This may well have been; but at the same time, there was sufficient authority on the other side to establish that some cases of arsenical poisoning had actually proved fatal in three or four hours. So far as this objection was concerned, the prisoner was very properly convicted. On the medical question raised at this trial, it may be observed, that two distinct cases have occurred in which the persons died certainly within two hours after they had taken this poison; and several instances have been reported since this trial, in which death took place in from three to four hours after the administration of arsenic. It seems extraordinary in the present day, that any attempt should have been made by a professional man to negative a charge of criminal poisoning upon so weak a ground as this; but we must remember that this opinion was expressed many years ago, when the subject of poisoning, in its medical relations, was but little understood. It is quite obvious that there is nothing, so far as we know, to prevent arsenic from destroying life in an hour. These matters can only be settled by a careful observation of numerous cases, and not by any *a priori* reasoning, or reference to a limited personal experience.

In all instances of *sudden death*, there is generally a strong tendency on the part of the vulgar to suspect poisoning. They never can be brought to consider that persons may die a natural death suddenly, as well as slowly; or, as we shall presently see, that death may really take place slowly, as in cases of disease, and yet be due to poison. This prejudice continually gives rise to the most unjust suspicions of poisoning; cases illustrative of this have already been given (*ante*, p. 83). One of the means recommended for distinguishing narcotic poisoning from apoplexy or disease of the heart, is the difference in the rapidity with which death takes place. Thus, apoplexy or disease of the heart may prove fatal either instantly or within an hour. The only common poison likely to operate with such fatal rapidity is prussic acid. But when this is the cause of death, some traces of the poison may be found at hand, except in a case of murder, when it has been intentionally removed. Poisoning by opium is commonly protracted for five or six hours. This poison has never been known to destroy life instantaneously, or in a few minutes. Thus, then, it may happen, that death will occur with such rapidity as to render it impossible to attribute it to narcotic poison.

Chronic Poisoning.—When a poison destroys life rapidly, it is called a case of *acute* poisoning, to distinguish it from the *chronic* form, *i. e.*, where death takes place slowly. Chronic poisoning is a subject which of late years, in reference to arsenic, antimony, and lead, has required medico-legal investigation. Most poisons are capable, when their effects are not rapidly manifested, either from the smallness of the dose or from frequent repetition, of slowly undermining the powers of life, and killing the patient by producing emaciation and exhaustion. This is sometimes observed in the action of arsenic and corrosive sublimate, but it has been remarked also in cases of poisoning by the mineral acids and caustic alkalies. Death is here an indirect consequence—stricture of the œsophagus is induced, or the lining membrane of the stomach is destroyed and the process of digestion impaired—a condition which leads to innutrition and death. The time at which these indirect effects will prove fatal, is of course liable to vary. A person has been known to die from a stricture of the gullet brought on by sulphuric acid, *eleven months* after the poison had been swallowed; and there is no reason to doubt that instances may occur of a still more protracted kind. In these cases of chronic poisoning, there is considerable difficulty in assigning death exclusively to the original action of the poison, since the habits of life of a person, a constitutional tendency to disease, and other circumstances, may have concurred to accelerate or produce a fatal result. To connect a stricture of the gullet with the act of poisoning by a mineral acid, it is necessary to show that there was no tendency to this disease before the acid was administered—that the symptoms appeared soon after the first effects of the poison went off—that these symptoms continued to be aggravated until the time of death; and that there was no other intervening cause to which death could reasonably be referred. These remarks apply equally to the secondary fatal effects of any poison, such, for instance, as the salivation occasionally induced by corrosive sublimate, and the exhaustion and depression which are caused by tartarized antimony, when the acute symptoms of poisoning by these substances have passed away.

Several cases have come before our tribunals in which the facts connected with this form of poisoning were of some importance. I allude to those of Miss Blandy, tried at Oxford in 1752, for the murder of her father by arsenic; and of a woman named Butterfield, tried at

Croydon in 1775, for the murder of a Mr. Scawen, by administering corrosive sublimate. Among cases of recent occurrence may be mentioned that of Mrs. Wooler (*Reg.* v. *Wooler*, Durham Winter Assizes, 1855), in which it was clearly proved that the deceased had been under the influence of arsenic, administered at intervals in repeated doses, for a period of about seven weeks before her death. She died from exhaustion and the secondary effects of the poison. In three other cases tartarized antimony was the poison selected. It was given in repeated doses, over different periods, and caused death, by the specific effects of poisoning in a chronic form. 1. The case of Ann Palmer (*Guy's Hosp. Rep.*, Oct. 1857.) 2. The case of McMullen (Liverpool Summer Assizes, 1856), in which a woman was tried and convicted for causing the death of her husband; and 3d. The case of *Reg.* v. *Hardman* (Lancaster Summer Assizes, 1857), in which a man was convicted of the murder of his wife. In most cases, criminals destroy life by administering poison in one large dose; but in the instances referred to, small doses were given at intervals, a fact which, in some of them, led to a medical doubt of the real cause of the symptoms. The case of Isabella Banks (*Reg.* v. *Smethurst*, Central Criminal Court, Aug. 1859) gave rise to a great difference of opinion respecting the cause of death. Drs. Julius and Bird, who attended the deceased throughout her illness of about a month's duration, the late Dr. Todd and myself, referred the symptoms and cause of death to chronic poisoning by antimony and arsenic, and in confirmation of this opinion, antimony was distinctly found by Dr. Odling and myself in the intestines after death. Arsenic was also found in an evacuation passed by the deceased three days before her death. The late Dr. Tyler Smith, Dr. Richardson, and others, who did not see the deceased, ignored the existence of antimony in the body, and referred the symptoms and appearances partly to pregnancy, and partly to a sudden attack of severe dysentery. The jury found the accused guilty, but upon the medical doubt thus raised in the public mind respecting the cause of death, the accused was subsequently discharged.

A similar question arose in *Reg.* v. *Winslow* (Liverpool Autumn Assizes, 1860). The prisoner was charged with the murder of a Mrs. James by administering to her small doses of antimony. The suspicions of Dr. Cameron, who attended deceased, were excited by the intermittent and violent nature of the vomiting, as well as by the extreme depression. Antimony was found in the urine and fæces by Dr. Edwards; and, after death, this substance was discovered, in small quantity, in the viscera, by Dr. Edwards, the late Dr. Miller, and myself. The deceased was at the time laboring under malignant disease of the cæcum, but it was alleged that the antimony had accelerated her death. The jury acquitted the accused. The examination of the body of the sister of deceased, as well as of two other members of the family, led to the discovery of antimony, also in small quantity, in the viscera of each; and from the nature of the symptoms preceding death, as well as the general healthiness of the organs, no doubt was entertained by the medical witnesses that all these persons, members of the same household, had died one after the other from the effects of antimony, administered at intervals in small doses. The law could not fix the perpetration of these four murders upon any person, and three would have wholly escaped public notice, but for the death of Mrs. James, some months after the bodies of the others had been buried under medical certificates setting forth natural causes of death! A set of cases somewhat similar has been brought to light by certain inquests on exhumed bodies at Bilston in Staffordshire (December, 1871). Three children in a family died at different times under similar symptoms. Dr. Hill, of Birmingham, found antimony in two of the bodies; and the body of a third child was exhumed after two months' burial, and antimony was also found in it. It appears that this child died on October 10th, and its death was registered on the 13th of that month as death from "asthenia," and "gastric fever," "six days!" They all received medical attendance, and their names, it is stated, were entered in some burial club! The occurrence of such cases as these suggests grave reflections on the insecurity of life, when poison is used with skill and cunning; and they further demonstrate the inefficiency of the present system of registering causes of death. (Modified by 37 and 38 Vic., cap. 88; August, 1874.) They show that medical men, in signing certificates, have not sufficiently inquired into the nature of the fatal illness, or the immediate cause of death (see *Lancet*, 1870, vol. 1, p. 341); but this is an evil which admits of a remedy. The public have much more to dread in the fact that, even in plain cases of poisoning, some physicians of experience and repute have been unable to discriminate the symptoms from those of natural disease..

The characters of chronic poisoning have a special interest for the medical jurist. There is one difficulty about them which no accuracy of observation or judgment can surmount. The poison or poisons, if found in the dead body at all, must necessarily exist in fractional parts of a grain. This alone will be sufficient to create a doubt whether death has been caused by the poison, although it is quite consistent with medical experience that a person may die from chronic poisoning, and little or none of the poison be found in the body after death. In the case of Mrs. James (*Reg.* v. *Winslow*), not more than the tenth part of a grain was found in the whole of the tissues of the body; in the case of Isabella Banks (*Reg.* v. *Smethurst*), the quantity was greater than this, but less than a grain altogether; while in the case of Mrs. Peters, of Yeovil, examined by the late Mr. Herapath, *none* was found in the body, although this chemist had extracted a quantity of antimony as sulphide from the urine of deceased, in less than nine days before her death! It has been already remarked that some poisons have what is called an *accumulative* property, *i. e.*, they may be administered for some time in small doses without producing any marked effects; but they will, perhaps, after a certain period, suddenly and unexpectedly give rise to violent symptoms affecting the life of a person. This peculiar mode of action has been witnessed more in medical practice than in cases of attempts to poison; hence it is not a subject of much importance to a medical jurist. Foxglove (digitalis) is said to possess this property. It has been remarked, on more than one occasion, that persons to whom this medicine had been repeatedly administered in medicinal doses have died suddenly, probably from the accumulative properties of the poison. The same effect has been noticed in the use of the hydrate of chloral and of chloroform vapor.

This accumulative property is probably dependent on the retention of the poison in the blood. It is not eliminated by the lungs or excretions with sufficient rapidity, and it is not deposited in the organs. The point of saturation of the blood varies, it is believed, with each poison, and ranges within small limits. The rate of elimination is also known to be variable. These facts may serve to explain why it is that an accumulative property is sometimes manifested. The dose may not have been increased at all, or but very slightly, and the blood may retain so much of the poison that a very small addition may render it fatal. In the operation of some poisons through the lungs, *e. g.*, of the diluted vapor of chloroform, it has been observed that dangerous consequences may ensue if a maximum effect is produced under continued respiration. It has been found better to withdraw the vapor before the full effect is produced, as the insensibility increases or deepens even after the removal of the vapor. The maximum effect of the poison on the brain and nervous system is not at once induced.

2. *Evidence from Appearances after Death.*—One of the principal means of determining whether a person has died from poison is an examination of the dead body. In relation to *external appearances*, there are none indicative of poisoning upon which we can safely rely. The body cools and becomes rigid as in death from natural causes. In death from strychnia, the rigidity has been observed to supervene rapidly and to remain for an unusual time. As to the rate of cooling, no difference has been noticed, when all the circumstances connected with the cooling of the body have been duly considered.

At an inquest which occurred at West Haddon, in Northamptonshire, in January, 1874, the question was whether an aged lady had died from disease of the heart under which she was laboring, or from a volatile poison, such as prussic acid, given to her shortly before death. A chemical expert, who gave evidence on this occasion, laid great stress on the retention of warmth by the dead body as evidence of this form of poisoning. Such a condition, however, is not a characteristic of poisoning with prussic acid. It may equally occur in death from disease, so that it would furnish no criterion whatever of the cause. (*Guy's Hosp. Reports*, 1874, p. 467.) In this case, however, the warmth of the body was sufficiently explained by the sudden death of the deceased, and by the simple fact that there had not been time for it to cool! A medical man, who made merely a manual observation of the warmth, saw the body only a quarter of an hour after death, and the body was well covered with blankets and bedclothes. It was formerly supposed that the bodies of persons who were poisoned, putrefied more readily than those of others who had died from natural disease; and evidence for or against poisoning was at one time drawn from the external appearance. This is now known to be an error; the bodies of persons poisoned are not more rapidly decomposed, *cœteris paribus*, than those of others who have died a sudden and violent death from any cause whatever.

Irritant poisons act chiefly upon the stomach and intestines, which they irritate, inflame, and corrode. We may likewise meet with all the consequences of inflammation, such as ulceration, perforation, and gangrene. Sometimes the coats of the viscera are thickened, at other times thinned and softened by the action of an irritant.

Neurotic poisons—those which have a cerebral action and the true spinal poisons (strychnia and brucia), do not commonly leave any well-marked appearances. The stomach and intestines present no unnatural changes. There is greater or less fulness of the bloodvessels of the brain or its membranes; but even this is often so slight as to escape notice, unless attention is particularly directed to the brain. An effusion of blood is rarely found. Some of the neurotic poisons which affect both the brain and spinal marrow (aconite) may produce congestion of the brain or of its membranes, as well as marks of irritation in the stomach and bowels, and commonly both, according to their peculiar mode of action.

The *Spinal* poisons, in the form of alkaloids, present no well-marked or uniform appearances in the dead body.

It is important to bear in mind, that both irritant and neurotic poisons may destroy life without leaving any appreciable changes in the body. To such cases as these, the remarks about to be made do not apply. The proofs of poisoning must, then, be obtained from other sources. Any evidence derivable from appearances in the body of a person poisoned, will be imperfect unless we are able to distinguish them from those analogous changes, often met with as the results of ordinary disease. These are confined to the mucous membrane of the stomach and bowels. They are redness, ulceration, softening, and perforation. Any of these conditions may depend upon disease, as well as upon the action of irritant poisons.

Redness.—It is a main character of the irritants to produce redness of the mucous membrane of the stomach and small intestines. This redness, when first seen, is usually of a deep crimson color, becoming brighter by exposure to air. It may be diffused over the whole mucous membrane—at other times it is seen in patches, dots, or streaks (striæ), over the surface of the stomach. It is sometimes met with at the smaller, but more commonly at the larger end of this organ; and again, we occasionally find that the folds or prominences only of the mucous membrane present a red or inflamed appearance.

Redness of the mucous membrane may, however, be due to gastritis or gastro-enteritis (*ante*, p. 97); and in order to assign the true cause, it will be necessary to have an account of the symptoms preceding death, or some proof of the existence of irritant poison in the contents of the stomach or the tissues of the body. In this respect the case, *Reg.* v. *Hunter*, Liverpool Spring Assizes, 1843, is of some importance. A woman was charged with having poisoned her husband with arsenic. The medical evidence rested on the symptoms as well as on the appearances after death, for no arsenic was discovered in the body. The mucous membrane of the stomach and intestines was found, throughout its whole extent, much inflamed and softened. The medical witnesses for the prosecution referred this condition to the action of arsenic;

those for the defence considered that it might be owing to idiopathic (spontaneous) gastro-enteritis, independently of the administration of any irritant substance. The circumstances of the case were very suspicious; but the prisoner was acquitted, not merely on account of the variance in the medical evidence, but from the absence of positive proof of the presence of poison in the body, *i. e.*, its detection by chemical analysis (see *ante*, p. 42, also the published reports of the case by Mr. Holland and Mr. Dyson).

In the healthy state, the mucous membrane of the stomach is pale and white, or nearly so, except during digestion, when it becomes reddened; and some observers have remarked that a slight redness has often remained in the stomachs of those who have died during the performance of the digestive process. When in contact with the spleen or liver in the dead body, the stomach is apt to acquire a deep livid color from the transudation of blood; and it is well known that the bowels acquire a somewhat similar color from the gravitation of blood, which always takes place after death. None of these appearances are likely to be mistaken for the action of an irritant poison.

There is an important class of cases in which redness of the mucous membrane of the stomach is found after death, and is not dependent on the action of poison or on any assignable cause. These cases, owing to their being so little known, and involved in much obscurity, deserve great attention from a medical jurist; since the appearances closely resemble those produced by irritant poison. A person may die without suffering from any symptoms of disordered stomach; but, on an inspection of the body, a general redness of the mucous membrane of this organ will be found, not distinguishable from the redness which is so commonly seen in arsenical poisoning. Several cases of this kind have occurred at Guy's Hospital; and drawings which have been made of the appearances presented by the stomach are preserved in the Museum collection. A record has been kept of four of these; and it is remarkable that, although in not one of them, before death, were any symptoms observed indicative of irritation or disease of the stomach, yet in all, the stomach was found more or less reddened, and in two extensively so. Such cases are not very common; but the certainty of their having occurred where poisoning could not be suspected, should place the witness on his guard, so that he may not be led to countenance a suspicion of poisoning too hastily. In order to distinguish them, we must note whether there have been any symptoms during life, and their nature; as in the above cases, there may have been no symptoms, or they may have amounted only to slight disturbance of the stomach. Under these circumstances, they could not be mistaken for symptoms of irritant poisoning. Such cases are only likely to lead into error those who trust to this appearance alone as evidence of poisoning; but no medical jurist, aware of his duty, could ever be so misled.

This pseudo-morbid redness of the stomach may truly occur where there is some ground for suspicion, as in the following case: A young woman, far advanced in pregnancy, died suddenly in a fainting fit, one morning soon after she had risen. She had been in ill health previously; but there was nothing to indicate that she had taken poison;

indeed, from what has been already said, the suddenness of her death was rather adverse to the suspicion that she had died from such a cause. Yet after death it was found, among other appearances, that the mucous membrane of the stomach was inflamed (reddened?) and raised in folds. There had been no symptoms of irritation in the stomach or bowels. A case, in which it is probable this pseudo-morbid appearance of the mucous membrane was mistaken for the effects of irritant poison, will be found in the *Ann. d'Hyg.*, 1835, vol. 1, p. 227.

Dr. Yelloly long since remarked, that the mucous membrane of the stomach often presented a high degree of vascularity (redness) in cases of sudden death. He met with this appearance in the stomachs of some executed criminals, whose bodies were examined soon after they had undergone the sentence of the law. From his observations it appears—1. That vascular fulness of the lining membrane of the stomach, whether florid or dark-colored, is not a special mark of disease, because it is not inconsistent with a previous state of perfect health. 2. That those pathologists were deceived, who supposed from the existence of this redness in the stomach, that gastritis sometimes occurred without symptoms. 3. That erroneous conclusions on the cause of death were frequently owing to similar mistaken observations—the effects of putrefaction and spontaneous changes, induced by the loss of vitality, being sometimes attributed to the action of poisons. 4. That the redness in question is entirely venous, the florid state of the vessels arising from the arterial character of the blood remaining in the veins for some time after its transmission from the arterial capillaries at the close of life—the appearance is, however, sometimes due to transudation only. 5. That the fact of inflammation having existed previously to death, cannot be inferred merely from the aspect of the vessels in a dead part—there must at least have been symptoms during life. (See *Medical Gazette*, vol. 17, p. 309.) Andral and other pathologists have adopted similar views, and these views have obviously an important bearing upon medico-legal practice, since there is generally a tendency to suspect poisoning whenever redness of the mucous membrane of the stomach is met with in the dead. Such a condition does not even prove the past existence of inflammation, unless there were symptoms during life or other marked effects of the inflammatory process in the alimentary canal. It can be no sign of poisoning, unless the presumption is supported by evidence from symptoms, or by the actual discovery of poison in the stomach or other viscera.

In the case of Good, executed some years since, the mucous membrane of the stomach was found reddened, as if from the action of an irritant poison; and, in one instance of death from asphyxia by carbonic acid, there was not only reddening but effusion of blood in the stomach. The deceased was found dead in her bed, and the husband was suspected of having destroyed her by poison. A minute investigation of this case, made by Sir James Paget and myself, showed that the suspicion was unfounded. The woman had died from the effects of carbonic acid from the flue of a coke stove.

The redness in the stomach, in cases of poisoning, is so speedily altered by putrefaction, when circumstances are favorable to this process, as to render it difficult for a witness to speak with any certainty upon its cause. Putrefactive infiltration from the blood contained in the adjacent viscera and muscles, will give a reddish-colored appearance to a stomach otherwise in a healthy condition. Great dispute has arisen respecting the length of time during which redness of the stomach produced by an irritant will be recognizable and easily distinguishable from putrefactive changes. It is, perhaps, sufficient to say that no certain rule can be laid down on this subject: it must be left to the knowledge and discretion of the witness. It will depend on the nature of the poison taken and the degree of putrefaction. I have distinctly seen the well-marked appearances of inflammation produced by arsenic in the stomach and duodenum, in an exhumed body, twenty-eight days after interment (*Reg.* v. *Jennings*, Berks Lent Ass., 1845); and in another instance (August, 1846) the reddened state of the mucous membrane, in a case of arsenical poisoning, was plainly perceptible on removing a layer of arsenic *nineteen months* after interment. In the case of Mrs. Bacon the redness of the stomach and bowels was apparent, although the body had been *twenty-one months* in the grave. (*Reg.* v. *Bacon*, Lincoln Summer Ass., 1857.) (See on this question, a case of suspected poisoning by Orfila, *Annales d'Hyg.*, 1839, vol. 1, p. 127.) If, however, there be a reasonable doubt respecting the cause of the redness, it would be proper not to rely upon it as evidence of poisoning.

Ulceration.—In irritant poisoning the stomach is occasionally found ulcerated; this is, comparatively speaking, a rare occurrence in acute cases, but not unfrequent in chronic or protracted cases of poisoning with arsenic. In these cases the mucous membrane is removed in small distinct circular patches, under the edges of which the poison (arsenic) is often found lodged. Ulceration of the stomach is perhaps a more common result of disease than of the action of poison. As a consequence of disease, it is very insidious, going on often for weeks together, without giving any indications of its existence, except perhaps slight disturbance of the stomach, with occasional nausea, vomiting, and loss of appetite. In this case the ulceration is commonly seen in small circumscribed patches. It is worthy of remark, as one means of distinction, that ulceration has never been known to take place from arsenic or any irritant poison until *after* symptoms indicative of irritant poisoning have occurred. In ulceration from disease the mucous membrane is commonly only congested in the neighborhood of the ulcer. In ulceration from poison the redness is generally diffused over other parts of the stomach, as well as over the duodenum and small intestines. A case, however, occurred in Guy's Hospital, some years ago, in which, with a small circular patch of ulceration near the cardiac opening, the whole mucous membrane was red and injected; but this singular condition of the stomach, so closely resembling the effects of an irritant poison, was unaccompanied by any marked symptoms during life. The history of a case previous to death will thus commonly enable us to determine to what cause the ulceration found may be due. In this disease death may take place after a few hours' illness, and thus simulate poisoning, for the attack may commence soon after a meal. Dr. Procter, of York, communicated to me the following case (March, 1873): A man, æt. 63, in his usual state of health, was suddenly seized with a violent pain in his stomach about two hours after his breakfast. He took a few drops of laudanum, vomited it, but received no relief. He soon fell into a state of collapse, and died in two hours from his first attack. He did not vomit much, but complained of nausea. On inspection, the chief morbid appearances were in the stomach. The whole of the mucous membrane was congested and somewhat softened, especially at the intestinal end. At this part there were five small ulcers with smooth edges, not perforating the stomach. The contents yielded no trace of poison. The walls of the heart were thin and the mitral valves thickened.

During life this man had shown no symptoms indicative of acute or chronic disease of the stomach. The sudden access of pain—the absence of vomiting and purging, and the absence of poison, would have sufficiently distinguished this case from one of irritant poisoning.

Care must be taken to distinguish ulceration from corrosion. Ulceration is a vital process; the substance of a part is removed by the absorbents as a simple result of inflammation. Corrosion, on the other hand, is a chemical action—the parts are removed by the immediate contact of the poison; they are decomposed; their vitality is destroyed, and they combine with the corrosive matter itself. Ulceration requires time for its establishment, while corrosion is generally an instantaneous effect.

There is no form of acute poisoning in which a medical jurist has it more in his power to pronounce an opinion from appearances in the dead body, than in cases of death from the mineral acids. Tartra long since observed that whenever the alimentary canal, from the mouth to the intestines, was found corroded and converted into a soft fatty substance of a bright yellow or brown color, when it was easily detached from the subjacent parts, and there were marks of inflammation or gangrene or actual perforation of the stomach, there could be no doubt that these effects were due to a mineral acid, whether the acids were discovered in the body or not. Sir R. Christison has also adopted this view; and he very properly remarks that such cases must be considered as distinct exceptions to the general rule, regarding the weakness of evidence derived from appearances after death. Indeed, it may be inquired of those who are disposed to entertain an adverse opinion, what conceivable form of disease can produce such well-marked appearances simultaneously in the mouth, throat, gullet, stomach, and intestines, in the course of a few hours?

Softening.—The coats of the stomach are not unfrequently found so soft, as to break down under very slight pressure; and this may be the result either of poisoning, of some spontaneous morbid change in its structure during life, or of the solvent action of the gastric juice after death. As this change in the stomach, when caused by poison, is commonly produced by those substances only which possess corrosive properties, it follows that in such cases, traces of their action will be perceived in the mouth, throat, and gullet. In softening from disease, the change will be confined to the stomach alone, and it is commonly found only at the cardiac extremity of the organ. When softening is really caused by an irritant poison, it is generally attended by other striking and unambiguous marks of its operation. Softening is not to be regarded as a common character of poisoning: it is only an occasional appearance. In one case of arsenical poisoning, the coats of the stomach were thickened almost of a gelatinous consistency, and free from any redness as a result of inflammation. I have met with a case in which the coats of the stomach were considerably hardened by sulphuric acid, and in one instance, hardened and thickened by arsenic. Softening can never be inferred to have proceeded from poison, unless other well-marked changes are present, or unless the poison be discovered in the softened parts. The stomachs of infants have been frequently found softened from natural causes—such cases could not be mistaken for poisoning, since the history during life—the absence of symptoms and of other appearances indicative of poisoning, as well as the total absence of poison from the viscera, would prevent such a suspicion from being entertained.

CHAPTER XVII.

EVIDENCE OF POISONING IN THE DEAD BODY—PERFORATION OF THE STOMACH—SPONTANEOUS PERFORATION—GASTRIC EROSION—PERFORATION OF THE ŒSOPHAGUS AND THE INTESTINES—PERFORATION BY FOREIGN BODIES—BY WORMS.

Perforation of the Stomach.—The stomach may be found perforated either as a result of poisoning or disease.

1. *Perforation from Poisoning.*—This may occur: 1. By corrosion. 2. By ulceration. The perforation by *corrosion* is yb far the most common variety of perforation from poisoning. It is occasionally witnessed when the strong mineral acids have been taken, especially sulphuric acid; the stomach, in such cases, is blackened and extensively destroyed; the aperture is large, the edges are rough and irregular, and the coats are easily lacerated. The poison escapes into the abdomen, and may be readily detected by chemical analysis. The perforation from *ulceration*, caused by irritant poison (arsenic), is but little known. There are, so far as I know, but few instances on record. In a great number of poisoned subjects examined during many years past at Guy's Hospital, not a single case had occurred. It must then be looked upon as a rare appearance in cases of irritant poisoning. In fact, the person dies from the specific effects of the poison, before there is time for perforation by ulceration to take place.

2. *Perforation from Disease.*—This is by no means an unusual occurrence. Many cases of this description will be found reported elsewhere. (*Guy's Hosp. Rep.*, No. 8.) It is invariably fatal when it proceeds so

far that the contents of the stomach escape into the abdomen; but sometimes the stomach adheres to the pancreas during the ulcerative process and then the person may recover.

The *symptoms* from perforation commonly show themselves suddenly, while the person is apparently in good health. Such cases may be easily mistaken for irritant poisoning. The principal facts observed with regard to this formidable disease are the following: 1. It often attacks young women from eighteen to twenty-three years of age. 2. The preceding illness is slight; sometimes there is merely loss of appetite, or capricious appetite, with uneasiness after eating. 3. The attack commences with a sudden and most severe pain in the abdomen, generally soon after a meal. In irritant poisoning, the pain usually comes on gradually, and slowly increases in severity. 4. Vomiting, if it exist at all, is commonly slight, and is chiefly confined to what is swallowed. There is no purging; the bowels are generally constipated. In irritant poisoning, the vomiting is usually severe, and purging seldom absent. 5. The person dies commonly in from eighteen to thirty-six hours—this is also the average period of death in the most common form of irritant poisoning, *i. e.*, by arsenic; but in no case yet recorded, has arsenic caused perforation of the stomach within twenty-four hours; and it appears probable that a considerable time must elapse before such an effect could be produced by this or any irritant. 6. In the perforation from disease, the symptoms and death are clearly referable to peritonitis. The aperture is commonly of an oval or rounded form, about half an inch in diameter, situated in or near the lesser curvature of the stomach, and the edges are smooth. The outer margin of the aperture is often blackened, and the aperture itself is funnel-shaped from within outwards, *i. e.*, the mucous coat is the most removed, and the outer or peritoneal coat, the least. The coats of the stomach, round the edge of the aperture, are usually thickened for some distance; and when cut, they have almost a cartilaginous hardness. These characters of the aperture will not alone indicate whether it is the result of poisoning or disease; but the absence of poison from the stomach, with the want of other characteristic marks of irritant poisoning, would enable us to say that disease was the cause. Besides, the history of the case during life would materially assist us in our judgment. The great risk in all these cases is, that the effects of disease may be mistaken for those of poisoning; for we are not likely to mistake a perforation caused by irritant poison for the result of disease.

Among numerous instances, tending to show the medico-legal bearings of this subject, I shall select one, which came before Mr. Hilton and myself for examination. A young lady, in a noble family, æt. 23, died somewhat suddenly, under suspicious circumstances. She had been unwell for about three weeks, and was subject to occasional vomiting and disorder of the stomach. Still, her illness was so slight that it did not in the least interfere with the performance of her usual duties. One afternoon, about four o'clock, and about three hours after her last meal, she was suddenly seized with the most excruciating pain in the abdomen, and violent vomiting. Her skin was cold and clammy, and the abdomen tender and painful. It was suspected that she had taken poison; and magnesia and sulphate of magnesia were given to her. No poison was found in the room, and she strongly denied the imputation. The symptoms became worse, the vomiting more violent, and she died the following morning, about fifteen hours after her first seizure. On inspection, all the organs were found healthy, except those of the abdomen. There were here strong marks of peritoneal inflammation; the intestines were loosely adherent to each other, and a quantity of lymph was effused around them. The cavity contained about a pint of liquid, which had escaped from an aperture in the stomach. The liquid was reserved for analysis. The stomach was laid open by making an incision along its greater curvature. It was empty. At the upper and posterior part, near the pyloric end of the smaller curvature, was an opening of an oval shape, about half an inch in its longest diameter. The edges were firm, hard, and smooth, presenting not the least appearance of laceration or ulceration. They were bevelled off from within outwards, being thinned towards the peritoneal coat, the aperture in which was much smaller than that in the mucous membrane. There was no sign of inflammation in the membranes around; but the peritoneum, about the edge of the aperture, had a dark appearance, and the coats of the stomach were thickened. At the lower part, near the larger curvature, there were thick, irregular lines of blackness (striæ), the mucous membrane being raised and blackened, but not softened. These black lines appeared like those produced by sulphuric acid; but there was no corrosion, and on applying test-paper there was no acid reaction. The black matter was interspersed with a yellowish-colored substance. The liquids taken from the abdomen, as well as the coats of the stomach, were chemically examined; but not a trace of poison could be detected. Considering the time of the occurrence of symptoms, their nature, and the absence of poison from the viscera and their contents—the suspicion of poisoning was at once negatived, especially when the above facts were taken together with the appearances in the dead body. The medical opinion given was: 1. That the deceased had died from peritonitis, caused by the escape of the contents of the stomach. 2. That this escape was owing to a perforation of the coats of the organ, caused by slow and insidious disease, and not by poison. (See *Guy's Hospital Reports*, October, 1850, p. 226.) In another suspected case, the body was exhumed after several months' burial, and on examination, the stomach was found perforated in the usual situation. There was no poison.

It has been hitherto supposed that perforation of the stomach must necessarily prove fatal. This is undoubtedly the ordinary result, but the fatal effect depends on peritonitis, excited by effusion of the contents of the organ. Under favorable circumstances, and by judicious treatment, no effusion may take place, and the person may entirely recover—as the aperture is ultimately closed by adhesion to the surrounding viscera. Of this closure of ulcerated apertures in the stomach, several specimens are preserved in the Museum of Guy's Hospital. This fact is of importance to the medical jurist, as a case might, from the symptoms, be mistaken for one of poisoning; although, when taken on the whole, they are unlike those produced by irritant poison. An apparently well-marked instance of recovery from perforation is reported by the late Dr. Hughes. (*G. H. Rep.*, N. S., vol. 4, p. 332.) The patient recovered from the first attack, but ultimately died from another perforating ulcer, which led to extravasation. (See also *Case by Mr. Hilton, ib.*, p. 343.)

Spontaneous or Gelatinized Perforation.—The stomach is occasionally subject to a spontaneous change, by which its coats are softened and give way, generally at the larger end. As the effusion of the contents of the organ in such a case never gives rise to peritoneal inflammation, and no symptoms occur prior to death to indicate the existence of so extensive a destruction of parts, it is presumed that the stomach undergoes a process of solution soon after death. It is commonly attributed to the solvent action of the gastric juice—the spleen, diaphragm, and other viscera in contact with the stomach being sometimes softened. My colleague, Dr. Wilks, who for many years conducted the inspections at Guy's Hospital, informed me that this post-mortem or cadaveric perforation of the stomach was so rare a condition that it was not met with more than once in five hundred cases. In two cases in which it was observed, one patient had died from albuminuria and the other from head-affection; but in neither of these could there be found any peculiarities regarding their food, the time of the last meal, or the state of the bodies to account for the spontaneous destruction of the coats of the stomach. (For some remarks on this subject, by Dr. Budd, see *Med. Gaz.*, vol. 30, p. 895; and by Dr. Pavy, on *Gastric Erosion*, see *G. H. Rep.*, 1868, p. 494.) In January, 1845, I met with an instance of spontaneous perforation in a child between two and three years of age. It was seized with convulsions, became insensible, and died in twenty-three hours. After death, the cardiac end of the stomach was found destroyed to the extent of three inches; and the edges were softened and blackened. There was no food in the stomach,, nor had anything passed into the organ for thirty-two hours before death! It was therefore impossible to ascribe death to the perforation, or the perforation to poison. (For a full account of this case, see *Med. Gaz.*, vol. 36, p. 32.) In October, 1846, I found a similar condition of the stomach in an infant aged nineteen months, suspected to have died from poison administered to it three months before. The cause of death in this case was mesenteric disease. The stomachs of children at the larger end are always very thin, and are often found quite pulpy on inspection, irrespective of disease or the presence of poison.

This form of perforation is reported to have been met with in the bodies of children affected with water on the brain—in those who have died from typhus fever—and, according to Andral, in women who have died during parturition. The late Dr. Macintyre informed me that he had met with two cases of this kind of perforation in young subjects affected with diabetes. The conditions for its production, whether local or constitutional, and the circumstances under which it occurs, are very obscure. (*Med. Gaz.*, vol. 39, p. 897; also vol. 41, p. 293.) The fact of most importance to a medical jurist is, however, that it is unattended by any marked symptoms during life. Some

French pathologists describe cases of what they term *gelatinized* perforation, in which disorder of the stomach had previously existed. Chaussier, indeed, believed that this form of perforation always depended on a particular disease of the organ; and he denied, from the results of his own experiments, that the gastric juice had any solvent action. (Flandin, *Traité des Poisons*, vol. 1, p. 259.) The inspection of the body, with a general history of the case, will, however, suffice to remove any difficulty in forming an opinion whether the extensive destruction commonly met with has or has not arisen from poison. Thus, in a cadaveric perforation, the aperture, which is always situated in that part of the stomach which lies to the left of the opening of the œsophagus, is very large, of an irregular form, and ragged and pulpy at the edges. These present an appearance as if they had been scraped. The mucous membrane of the stomach is not found inflamed. There is occasionally slight redness, with dark brown or almost black lines or streaks (striæ), in and near the dissolved coats, which have an acid reaction. It can only be confounded with perforation by the action of corrosives; but the well-marked symptoms during life, and the detection of the poison after death, together with the changes in the throat and gullet, will at once indicate the perforation produced by corrosive poison. A case of extensive perforation of the stomach as the result of the action of the gastric fluids, has been reported by Dr. Barnes. (*Med. Gaz.*, vol. 41, p. 293.)

The only case in which any mistake is likely to occur, is where, conjoined with the discovery of perforation after death, there may have existed some symptoms of irritation in the stomach and bowels during life. It is possible that a person may die under symptoms somewhat resembling irritant poisoning, and after death the gastric secretion may destroy the coats of the stomach; but such a singular combination of circumstances must be most unusual. This, however, signifies little in a legal point of view, for persons charged with the crime of poisoning, are frequently acquitted on the barest medical possibilities. One case of this doubtful character is on record. I allude to that of Miss Burns, for the murder of whom, by poison, a Mr. Angus, of Liverpool, was tried in the year 1808. It is not necessary to enter into the particulars of the case; since the appearances in the body are imperfectly described in the report. Although the symptoms, resembling irritant poisoning, under which the deceased labored, were not accounted for, yet there was great reason to believe that they were not connected with the perforation of the stomach, which, on the whole, bore the characters assigned to that produced by the gastric secretion after death. The charge of poisoning was not sustained either by the chemical or the pathological evidence, and the prisoner was acquitted. The evidence given on this trial is well worthy of the attention of every medical practitioner. It shows on what a nice balance of proofs charges of poisoning sometimes rest, and how important it is that a medical jurist should make himself acquainted with all the circumstances under which perforations of the stomach may occur.

Perforations of the Œsophagus (*gullet*) *and Intestines.*—Other parts of the alimentary canal are liable to become perforated; but not, so far as I have been able to ascertain, by the action of irritant poison. The gullet may become softened and destroyed by the contact of corrosive poison, but in general this rapidly passes through the tube and lodges in the stomach. There is one instance on record in which the gullet was completely destroyed by the action of sulphuric acid. As Sir R. Christison observes, it is not probable that a corrosive poison could ever perforate the intestines from within outwards, since its action would be chiefly expended on the stomach, and it is not likely to reach any portion of the intestines in a state sufficiently concentrated to destroy their coats by chemical action. (*On Poisons*, p. 149.) If a large quantity of corrosive poison flowed through an aperture in the stomach upon the intestines, then the coats might become destroyed from without inwards. The lower portion of the œsophagus, and various parts of the intestinal tube, have been found in several instances softened and destroyed, the aperture presenting all those characters which have been described in speaking of spontaneous or gelatinized perforation of the stomach. This change in the œsophagus is ascribed to a solvent action of the gastric juice, which enters the tube by regurgitation; but such an explanation cannot apply to the intestines. A case of softening of the intestines, from the duodenum to the sigmoid flexure of the colon, is reported by Mr. J. Smith: the child died of water on the brain. (See *Med. Gaz.*, vol. 2, p. 619.) The intestinal tube may become perforated in any part by ulceration, either as a result of disease or of the action of an irritant poison; but ulceration of the intestines from poison proceeding to a perforation of the coats, is a very rare condition.

Cases of *perforation of the intestines from disease* are occasionally met with. They require the attention of a medical jurist, since they may be easily mistaken for cases of poisoning. The following instance of perforation of the duodenum is reported by Mr. Bailey (*Med. Times*, Dec. 19, 1846, p. 223). A woman, æt. 28, was taken suddenly ill. There was great anxiety of countenance, with small and frequent pulse, cold extremities, vomiting, and occasional eructations of wind. She suffered severe pain in the region of the small intestines (duodenum), which caused her body to be doubled up. Her bowels had been only partially opened the day before. In about nine hours there was great tenderness of the abdomen; the pulse was smaller; but the bowels, in spite of the administration of medicine, were still unmoved. She gradually sank, and died in about fourteen hours and a half from the time she was first seized. Thus far the symptoms would indicate that the case was one of acute peritonitis and not of irritant poisoning. Nevertheless, the circumstances were extraordinary, and rumors were spread that her husband had poisoned her. The deceased, it appeared, had been married only the previous day; she and her husband did not retire to rest until three o'clock in the morning, and the attack came on suddenly, by a fit of severe pain, at nine o'clock, *i. e.*, six hours after they had retired to rest. An inspection of the body showed all the usual marks of peritoneal inflammation. The duodenum, in its transverse portion, was found to have a circular opening in it, surrounded by a black margin externally; while internally the perforation seemed larger, its sides sloping off. The mucous membrane was softened for some distance around the ulceration, affording evidence of the existence of previous inflammation. The only symptom manifested before the attack was, that she had been for some time subject to a pain in her right side. (*Cormack's Ed. Jour.*, June, 1845, p. 445; and *Lancet*, July 18, 1846, p. 67.)

A case of perforation of the small intestines (ileum), in which two apertures were found, is reported by Dr. Zartman. (*Casper's Wochenschrift*, März 14, 1846.) Death took place in two days from inflammation of the peritoneum. One of the apertures was only the sixth of an inch in diameter. The edges of both were smooth, as if punched out.

Perforation by Foreign Bodies.—There is another insidious form in which perforation of the intestines may present itself, and cause fatal peritonitis. This is by the formation of an ulcerated aperture in the appendix of the cæcum (vermiformis cæci), of which two cases have been communicated to me by a former pupil, both occurring in young men. The perforation was produced in these instances by the pressure of a hard substance lodged in the extreme end. In one instance I analyzed this hard concretion, and found it to consist of inspissated mucus, biliary matter, and a large quantity of carbonate of lime. It was of an irregular form and structure, and about the size of a large pea. In both instances death was caused by peritonitis, produced by extravasation of the contents of the intestines, and the aperture was so small that it might have been easily overlooked. It is remarkable that the introduction into the appendix cæci of any foreign substance, as the pip of an apple, or a small bean, or cherry-stone, is liable to excite fatal inflammation, ulceration, or gangrene. A case of this kind has been reported by Mr. Nelson. (*Amer. Journ. Med. Sci.*, Feb. 1847, p. 258.) The fact is important in relation to the causes of sudden death.

Perforation by Worms.—It is now generally admitted that the various species of worms which infest the intestines of the human subject, may, in some rare cases, by irritating or even perforating the coats, give rise to symptoms which may be mistaken for those of poisoning. A writer on toxicology, M. Flandin, denies that perforation can ever take place from this cause (*Des Poisons*, vol. 1, p. 304, 1846); but as this denial is based on theory, while the statement which he impugns rests upon good authority, it is unnecessary to discuss this as an unsettled question. He supports his opinion by the authority of Rudolphi, who states that the entozoa have no organs capable of perforating the intestinal coats; by the view entertained by M. Dujardin, that worms are never injurious to animals, because they are often found in large numbers when the previous health of the animal has been uninjured; and by the statement of Andral, who, however, merely says that if worms have the power of perforating the intestines, the cases in which this happens are exceedingly rare, a conclusion in which most pathologists will entirely agree. Several instances are on record in which perforation has been thus caused, and the worm or worms have been found in the peritoneal cavity. A well-marked case of perforation of the small intestines (ileum) by an ascaris lumbricoides, leading to death from effusion and peritonitis, is reported by Mr. Kell. The opening through the intestine was of a circular form, and corresponded in magnitude with the size of the worm, which was eight inches long. The worm was found among the intestines, between the umbilicus and pubes. (*Med. Gaz.*, vol. ii, p. 650.) These worms may even perforate the abdominal parietes (see cases by Dr. Young, in the same volume, p. 748). Vogel says of the ascaris lumbricoides, that in certain cases it is capable of perforating the intestine, by thrusting asunder with its head the fibres of the intestinal coats. (*Pathological Anatomy*, Day's translation, p. 464.) When perforation takes place from this cause, it will be indicated by the discovery of the entozoon in the cavity of the peritoneum, or near to the aperture in the intestine (see a case in *Cormack's Ed. Jour.*, June, 1845, p. 447).

In alleged irritant poisoning, when worms are discovered in the intestinal canal, an attempt is not unfrequently made in the defence to refer the symptoms of irritation to the presence of these entozoa. If the symptoms of poisoning be well marked, and some of the poison be discovered in the body, such a defence must be a hopeless struggle against medical facts. This objection was taken to the evidence from symptoms in one case of arsenical poisoning; but the disturbance occasioned by worms is commonly slight, and is very rarely accompanied by severe vomiting and purging. A solitary instance is quoted by Christison, in which a child appears to have died under symptoms of narcotico-irritant poisoning as an effect of worms. Several hundred ascarides were found in the intestines, and thirteen in the stomach. (*On Poisons*, p. 133.) In two instances of arsenical poisoning, I found a large ascaris lumbricoides in the small intestines; and in another case, three of these worms, each from six to eight inches long, were found living in the duodenum a week after the death of the person, who had been poisoned. In the above cases there could not be the slightest doubt, in spite of the presence of the worms, that the sole cause of death was poison. In one instance the ascaris was well washed from adhering mucus, and examined for arsenic by Reinsch's process: the poison was abundantly discovered in it. (*G. H. Rep.*, N. S., vol. 4, p. 462.) In the third case the man had taken calomel shortly before death, and mercury was extracted from the body of the worm after it had been washed. Under such circumstances it would be impossible to refer the symptoms of irritation to worms. A case will be mentioned in a future chapter, in which, on examining the body of a person who had taken an ounce and a half of muriatic acid, the jejunum was found perforated by a lumbricus; but the acid was beyond all doubt the cause of death.

That, however, the presence of worms in a body may be made a ground of defence, is sufficiently proved by two cases reported by M. Flandin. A young man was charged with having poisoned his father with arsenic. A very small quantity of the (absorbed) poison was detected in the tissues of the body only. The report of the inspection stated that there was a large number of the ascarides lumbricoides in the intestinal canal, and some were even found in the œsophagus. A discussion on the cause of death arose between the counsel employed in the defence, who based his arguments on a memoir by M. Raspail, and the medical witness, who had discovered arsenic in the body as well as in the liver. It was contended that the quantity of arsenic found was not sufficient to cause death, and that the presence of the ascarides explained satisfactorily the cause of the symptoms, and their rapidly fatal course. The deceased, it was alleged, had been suffocated by the worms, which had caused the compression of the bowels, and had even ascended into the œsophagus! The witnesses replied that arsenic, even in small quantity, was never found in the healthy human body—that the presence of worms could not account for the presence of absorbed arsenic in the tissues—while the detection of the poison, on the other hand, satisfactorily accounted for all the symptoms, even for the passage of the worms into the œsophagus as a result of violent vomiting. To no other circumstance could death be attributed.

In the second case, an empiric applied to the cancerous breast of a woman, a plaster covered with white arsenic. Symptoms of poisoning by absorption followed, and the woman died. Arsenic was detected in the liver. An ascaris was found in the intestines. On removing the whole of the viscera, which were in a highly putrefied state, a portion gave way, and the worm appeared through the lacerated aperture. There had been no extravasation, nor any mark of peritonitis. The defence was, that the deceased had died from perforation of the intestine by the worm. This was denied by the witnesses, who contended that had this been the case, there would have been extravasation. The escape of the worm was owing to accidental laceration of the viscera during the inspection. The symptoms of the disease were clearly those of poisoning and not of peritonitis. (*Des Poisons*, vol. 1, pp. 307 and 507.)

CHAPTER XVIII.

INSPECTION OF THE DEAD BODY—POINTS TO BE OBSERVED—PRESENCE OF STRANGERS—EXHUMED BODIES—IDENTITY OF SUBSTANCES—PRESERVING ARTICLES FOR ANALYSIS—THE USE OF NOTES IN EVIDENCE—MEDICO-LEGAL REPORTS—RULES FOR DRAWING THEM UP.

Inspection of the Body.—In investigating a case of alleged poisoning, a medical man may not have seen the person while living. He will then be required to make an examination of the body. In addition to certain points which have been elsewhere noticed in reference to the living (*ante*, p. 93), he will have to direct his attention to those which follow: 1. The *exact time* of death if known: he may thus determine for how long a period the person has survived after having been first attacked with symptoms. 2. Observe the attitude and position of the body. 3. Observe the state of the dress. 4. Observe all surrounding objects. Any bottles, paper packets, weapons, or spilled liquids lying about, should be collected and preserved. 5. Collect any vomited matters near the deceased. Observe whether vomiting has taken place in the recumbent position or not. If the person has vomited in the erect or sitting position, the front of the dress will commonly be found covered with the vomited matters. 6. Note the external appearances of the body, and whether the surface be livid or pallid. 7. Note the state of the countenance. 8. Note all marks of violence on the person, or discomposure of the dress—marks of blood, etc. 9. Observe the presence or absence of warmth or coldness in the legs, arms, abdomen, mouth, or axillæ; if possible use a thermometer for this purpose. 10. The presence of rigidity or cadaveric spasm in the body. To give any value to the two last-mentioned characters, it is necessary to observe the nature of the floor on which the body is lying, and whether this be clothed or naked, young or old, fat or emaciated. All these conditions create a difference, in respect to the cooling of the body and the access of rigidity. 11. If found dead—When was the deceased last seen living, or known to have been alive? 12. Note all circumstances leading to a suspicion of suicide or murder. 13. The time after death at which the inspection is made. 14. Observe the state of the abdominal viscera. If the stomach and intestines are found inflamed, the seat of the inflammation should be exactly specified; also all marks of softening, ulceration, effusion of blood, corrosion or perforation. The stomach should be removed and placed in a separate vessel, ligatures being applied at the cardiac and pyloric ends. 15. The fluids of the stomach, if this organ is opened during the inspection, should be collected in a clean *graduated* glass vessel: notice *a* the quantity, *b* the odor tried by several persons, *c* the color, *d* acid or alkaline reaction, *e* presence of blood, mucus, or bile, *f* presence of any undigested food; and here it may be as well to observe, that the presence of farinaceous matters (bread) would be indicated by the addition of iodine water, if the contents were not alkaline; of sugar by the sugar test—of fat by heat; *g* other special characters, among which may be included the nature of the *sediment*, if any, after standing. In cases of poisoning by mineral substances, there may be found a black deposit (soot) or a blue coloring matter (smalt, ultramarine, or indigo) from arsenic; vermilion or Prussian blue from phosphorus, with portions of matches and fragments of wood and sulphur. Prussian blue is also used for coloring, in Battle's vermin-killer; this substance may therefore indicate poisoning with strychnia.

In a case of poisoning with arsenic (*Reg.* v. *Windsor*, York Winter Ass., 1845) Mr. Procter found in the stomach of deceased 58 grains of arsenic mixed with 18 grains of white sand. This discovery connected the accused with the act of administration. The prisoner had been employed to sweep a room, the floor of which had been washed with bug-poison (containing arsenic) and sand. She had thus obtained access to the poison, and administered it with the sand. In a case referred to me, the arsenic found in the stomach of the deceased was colored with artificial ultramarine. This served to identify it with a packet of arsenic found concealed, in which a similar coloring material had been used in place of indigo.

In cases of poisoning with vegetable substances, we may meet with seeds and fragments of roots and leaves. These foreign matters will require, for their identification, a microscopical and botanical, as well as a chemical examination.

The fluids of the stomach, when first removed, may present a well-marked *odor* indicative of prussic acid, oil of bitter almonds, nitrobenzole, ether, alcohol, chloroform, or carbolic acid. A measured portion should in this case be submitted to distillation and the distillate examined. Sometimes the liquid requires to be warmed, either by itself or with the addition of an acid or an alkali, before an odor is evolved. In this case the odor of opium, cyanide of potassium, conia and nicotina, will become perceptible. According to the result of this examination, a portion of the liquid may be distilled and the distillate tested. 16. The contents of the duodenum should be separately collected, ligatures being applied to it. 17. Observe the state of the large intestines, especially the rectum, as poison has been sometimes introduced into this portion of the bowels by injection, and note the condition of their contents. The discovery of hardened fæces would prove that purging had not existed recently before death. In one case, in which I was required to give evidence, this became a question of considerable importance. 18. The state of the mouth, windpipe, throat, and gullet—whether there are in these parts any foreign substances or marks of inflammation and corrosion. This will throw light on the question whether the poison swallowed was a local irritant or corrosive, and whether it had or had not a chemical action. It also removes any doubt which might arise respecting death by suffocation from mechanical causes. 19. The state of the lungs and heart; all morbid changes noted. 20. The state of the brain and spinal marrow. 21. The condition of the uterus, ovaries, and genital organs should be examined, as poison has been sometimes introduced into the system by the vagina. 22. The liver with the gall-bladder should be removed for a chemical examination. 23. The urinary bladder, with any fluid contained in it, should be removed and placed in a separate jar.

Such are the points to which, in the greater number of cases of suspected poisoning, a medical jurist should attend. By means of these data, as well as others (*ante*, p. 93), noted according to the particular case to which they are adapted, he will in general be enabled, without difficulty, to determine the probable time of death, the probable cause of death, and the actual means by which death was brought about. He may thereby have it in his power also to point out the dish or article of food which had contained the poison, if the case be one of poisoning; and to throw light upon any disputed question of suicide or murder in relation to the deceased. Many cases of poisoning are obscure, owing to these points not having been attended to in the first instance.

I have not considered it necessary to enter into any details respecting the mode of performing an inspection. This the practitioner will have acquired during his study of anatomy; and the only essential points in addition to those abovementioned, are—1. To examine all the important organs for marks of natural disease; and 2. To note down any unusual pathological appearances, or abnormal deviations; although they may at the time appear to have no bearing on the question of poisoning. It is useful to bear in mind on these occasions, that the body is inspected, not merely to show that the individual has died from poison, but to prove that he has *not* died from any *natural cause.* Medical practitioners commonly give their attention exclusively to the first point; while lawyers, who defend accused parties, very properly direct a most searching examination to the last-mentioned point, *i. e.*, the healthy or unhealthy state of those organs which are essential to life, and with which the poison has not probably come in contact. The most usual causes of sudden death commonly have their seat in the brain, the heart with its great vessels, and in the lungs. Marks of effusion of blood or serum, congestion, inflammation, suppuration, or a diseased condition of the valves of the heart, should be sought for and accurately noted, whatever may be the condition of the abdominal viscera, or of the parts which are specially affected by the poison. It has also been recommended in cases in which the cause of death is at all obscure, after the general inspection of the body, that the spinal marrow and its membranes should undergo a special examination.

No persons should be allowed to be present at an inspection in a medico-legal case, excepting those who are directly empowered by a coroner or magistrate to undertake it. The presence of strangers interferes with the proper performance of this responsible duty. For anything known to the examiner, some of these spectators may have a direct interest in defeating the objects of the inspection. In a French case of alleged poisoning, in order to defraud an insurance company, in which I was consulted many years since, some persons—not apparently connected with the case—were allowed to be present as spectators at the inspection of the body. It was conducted in the usual way, and the viscera for analysis were placed in separate jars well secured and labelled. The analysis was subsequently made by an eminent expert, to whom the jars properly secured and labelled were afterwards sent. On opening the jar labelled "the stomach," it was found empty. The organ and its contents must have been removed before the jar was secured, as the seals were unbroken. Inquiry led to no result, and as there was no evidence of the presence of poison, the case fell to the ground. There was reason to believe that some person present at the inspection had secretly removed the stomach from the jar and had made away with it. The inspector omitted to look into the jar just before it was finally secured.

In the case of Cook (*Reg.* v. *Palmer*, 1856), the presence of the prisoner at the examination of the body led to the following results: While the stomach was being laid bare, the prisoner pushed against the person making the inspection, quite *accidentally* as it was stated, and thus caused a wound in the stomach, which led to the escape of some of the contents into the abdomen. The stomach was immediately transferred to a jar, cut open, and the remainder of its contents effused amidst portions of the intestines and their contents. The jar was then covered with wet bladder and securely tied; but while the attention of the inspectors was for a few moments withdrawn, the prisoner was seen to cut through the bladder and invert the jar, obviously for the purpose of allowing the fluid portion of the contents to escape. When the jar was delivered to Dr. Rees and myself for analysis, the cut thus made in the bladder was visible, but this had been so stretched after the *accident*, as to serve again the purpose of covering the top of the jar. These manipulations of the prisoner, which were concealed from us at the time, necessarily placed very serious difficulties in the way of a correct analysis.

A person suspected or accused of the act of poisoning, should not be allowed to be present at the medico-legal inspection of a body. A coroner may allow the attendance of some one to represent the accused, and make notes of the manner in which it is conducted; but he should not be permitted to interfere in any way with the proceedings of those who have received the usual legal order for a post-mortem examination.

Exhumed Bodies.—Sometimes the inspection of a body is required to be made long after interment. So long as the coffin remains entire, there may be the expectation of discovering mineral and some alkaloidal poisons in the organs; but decomposition may have advanced so far as to destroy all pathological evidence. The inspection in such cases is commonly confined to the abdominal viscera. The stomach is often found so thinned and collapsed, that the anterior and posterior walls appear to form only one coat. This organ should be removed with the duodenum, and ligatures should be applied to each. The liver and the spleen should also be removed, in order that they may, if necessary, be separately analyzed. If poison is not found in one or more of these parts, it is not likely that it will be discovered in the body. It has been recommended that a portion of earth immediately above and below the coffin should be removed for analysis, as it may contain arsenic; but this appears to me to be an unnecessary piece of refinement when the coffin is entire, or when the parietes still cover the viscera. If decomposition has so far advanced as to have led to an admixture of earth with the viscera, and the poison is found in minute quantity in the tissues only, the source of the poison may be regarded as doubtful. The body of a deceased person, when exhumed, should be identified by some friend or relative, in the presence of the medical examiner. In one case of murder by poison, the evidence almost failed, owing to this precaution not having been taken.

It is important that the viscera taken from a body which has been long in the grave should be sealed up immediately. They should not be allowed to come in contact with any metal, nor with any surface except that of clean glass, porcelain, or wood. It has been recommended that they should be washed with chloride of lime, or placed in alcohol; but this is decidedly improper: the use of any preservative chemical liquid would not only embarrass the future analysis, but would render a special examination of an unused portion of the liquid necessary, the identity of which would have to be unequivocally established. Preservation from air in clean glass vessels, with well-fitted corks covered with skin, or, what is still better, sheet-caoutchouc, is all that is required in practice. There is no objection to the use of a small quantity of chloroform. The vapor of this liquid diffused through the vessel tends to retard putrefaction. The contents of a stomach, consisting of blood and mucus, have thus been preserved in an unchanged state for several months.

Identity of Substances.—It is necessary to observe, that all legal authorities rigorously insist upon proof being adduced of the *identity* of the vomited matters or other liquids taken from the body of a deceased person, when poisoning is suspected. Supposing that, during the examination, the stomach and viscera are removed from the body, they should never be placed on any surface, or in any vessel, until we have first ascertained that the surface or vessel is perfectly *clean*. If this point be not attended to, it will be in the power of counsel for the defence to raise a doubt in the minds of the jury whether the poisonous substance might not have been accidentally present in the vessel used. This may be regarded as a very remote presumption; but, nevertheless, it is upon technical objections of this kind that acquittals follow, in spite of the strongest presumptions of guilt. This is a question for which every medical witness should be prepared, whether he is giving his evidence at a coroner's inquest, or in a court of law. Many might feel disposed to regard matters of this kind as involving unnecessary nicety and care, but if they are neglected it is possible that a case may be at once stopped: so that the care subsequently bestowed upon a chemical analysis will be labor thrown away. The strength of the evidence is determined by its weakest point. Proof of the presence of poison in the contents of a stomach was once rejected at a trial for murder, because the fluids had been hastily thrown into a jar borrowed from a neighboring grocer's shop; and it could not be satisfactorily proved that the jar was clean and entirely free from traces of arsenic, in which the grocer dealt. When the life of a person is at stake, as in a charge of murder by poisoning, the slightest doubt is always very properly interpreted in favor of the accused.

Not only must *clean* vessels be used for receiving any liquid destined for subsequent chemical analysis, but care must be taken that the *identity* of a substance is preserved, or the most correct analysis afterwards made will be inadmissible as evidence. The suspected substance, when once placed in the hands of a medical man, should never be let out of his sight or custody. It should be kept sealed under his private seal, and locked up while in his possession in a closet to which no other person can have access. If he has once let the article out of his hands, and allowed it to pass through the hands of several other persons, then he complicates the evidence for the prosecution, by rendering it indispensable for these persons to state under what circumstances it was placed while in their possession. The exposure of a suspected substance on a table, or in a closet or room to which many have access, may be fatal to its legal identity; for the chemical evidence, so important in a criminal investigation, will probably be altogether rejected by the court. A case was tried on the Norfolk circuit, in which an analysis of certain matters vomited by a person poisoned with arsenic was not admitted as evidence against the prisoner, because the medical man had left them in the custody of two women, and these women had allowed the vessel containing the suspected liquid (which was subsequently proved to contain arsenic) to be exposed in a room open to the access of many persons. In another case, tried at the Old Bailey Sessions, the analysis of some suspected liquids was not allowed in evidence, because the practitioner, who lived in the country, and was unwilling to take the responsibility of analyzing them, had sent them up to town by a carrier to be examined by a London chemist. If closely sealed by a private seal, and this is observed by the receiver to be unbroken, before he proceeds to an analysis, this mode of transmission will not probably be objected to. All wrappers and packets with their seals should be preserved for production if required. When any article (*e. g.*, a stomach or other organ) is reserved for analysis, care should be taken to attach immediately to it, or to the vessel containing it, a parchment or wooden label, upon which are plainly written, in ink, the name of the deceased and the date of removal, including the day of the week and month. This is especially necessary when there are two or more articles for analysis. I have known the greatest inconvenience to result from the neglect of this simple precaution.

Preserving Articles for Analysis.—In removing viscera or liquids from the body, and reserving them for analysis, it is necessary to observe certain precautions. A clean vessel, with a wide mouth, should be selected; it should be only sufficiently large to hold the organ or liquid (the less air remaining in it the better); it should be secured by a closely-fitting clean cork, covered with fine skin or bladder. Another piece of skin should then be tied over the mouth, or, for this, sheet caoutchouc or gutta-percha may be substituted with advantage. It should lastly be covered with tinfoil, and a layer of white leather. In this way any loss by evaporation or decomposition is prevented, and the viscera may be preserved (in a cool place) for some time. If the mouth of the vessel be too wide for a cork, the other articles cannot be dispensed with. Paper only should not be used: I have known the appearances after death of the viscera of an infant, suspected to have died from poison, entirely destroyed by drying, from the evaporation which took place through the layers of paper with which the vessel in which they were contained was covered. In reference to volatile poisons, such as prussic acid, the liquids supposed to contain them should be placed in glass-stoppered bottles. A medical jurist should bear in mind that all these minor matters are likely to come out in evidence; and whatever is worth doing at all, is worth doing well. For reasons already stated, antiseptic chemical compounds should not be used. The addition of a small quantity of chloroform to the viscera will, without complicating the analysis, tend to preserve them.

The articles used for the preservation of viscera should be in all cases scrupulously examined. Some kinds of calico are dressed with arsenic and starch paste, and many kinds of wrapping-paper as well as wall papers are strongly impregnated with this poison. An observation made by Mr. Aickin, of Belfast, shows that this is not an unnecessary caution. This gentleman was engaged in examining the body of a child, in order to determine the cause of death. The organs were healthy, and as no sufficient cause presented itself, he removed the stomach, with a view of making an analysis of its contents. He was suddenly called away; and, to preserve the stomach, he wrapped it in a piece of paper (used for papering rooms), placing it on the uncolored side, and he locked it in a closet until the following day. Assisted by a friend, he then analyzed the contents, and found a trace of morphia with a large quantity of arsenic. As the symptoms from which the child had suffered were not those of poisoning with arsenic, and there were no appearances of the action of this substance on the body, he came to the conclusion that there must be some extraneous cause to account for its presence. He examined a portion of the wall-paper in which the stomach had been wrapped, and then found that that part of it which was colored yellow was tinted with sulphide of arsenic or orpiment! It was therefore evident, as orpiment contains white arsenic, that the stomach and its contents had imbibed a portion of the poison during the night. (*Lancet*, June 23, 1855, p. 632.) This satisfactorily accounted for the presence of arsenic, under circumstances which might have given rise to a false charge of murder. Nearly all wall-papers, having any tinge of green or golden yellow in them, and some of those which are red, contain arsenic, and this arsenic spreads by imbibition to other parts of the paper not so tinted. It would, of course, be proper to avoid in all cases the use of any wrapper having upon it mineral colors of any description. Mr. Aickin's case shows in a striking point of view the danger of trusting to chemical analysis alone. Unless we look to physiology and pathology, a most erroneous opinion may be expressed. Dr. Reese, V. S., reports a case in which the search for arsenic failed to detect that poison; but zinc in large quantity was found. It turned out that the stomach had been thrown into an old tin can which had been formerly used for holding zinc-paint.

Arsenic is sometimes found mixed with oxide of iron in ochreous deposits or soils. It is thus occasionally present in the soil of cemeteries, but in an insoluble form. Even in the fur deposited in tea-kettles, in which there is generally some oxide of iron, an insoluble compound of arsenic has been found. From about a pound and a half of the crust or fur of a vessel, used for boiling water, Otto obtained well-marked arsenical deposits. Pöllnitz has detected in the fur of kettles copper, lead, tin, and even antimony. Dr. Osborn, of Southampton, has confirmed Pöllnitz's conclusion, namely, that lead is present in an insoluble form in the deposits of kettles and boilers. (*Med. Times and Gazette*, Dec. 22, 1860, p. 608.) Otto discovered a much larger proportion of arsenic in the calcareous crust taken from a kitchen boiler. Ten ounces of this gave a deposit of arsenic in a glass tube, and by Marsh's process several stains on white porcelain. He thinks that, if a sufficient quantity is employed, arsenic will be found in the sediment of all spring and well-waters. (*Ausmittelung der Gifte*, 1856, p. 61.) I have found arsenic in the water of rivers used for the supply of towns, and have extracted a well-marked quantity from two ounces of the dried mud of the Thames, as well as from similar quantities of earth taken from three churchyards in the north of England. (*Guy's Hosp. Reports*, Oct. 1860. On Arsenic and Antimony.) These facts, if they prove anything, tend to show the extreme danger of placing reliance on minute chemical results, or *traces*, as they are called, in the absence of good physiological and pathological evidence.

The results of an analysis, in the shape of sublimates or precipitates, should be carefully preserved for evidence, distinctly labelled, in small glass tubes hermetically sealed. They can then, if asked for, but not otherwise, be produced for examination at the inquest or trial.

On the use of Notes.—It has already been recommended, as a rule in these criminal investigations, that a practitioner should make notes of what he observes in regard to symptoms, appearances after death, and the steps as well as results of a chemical analysis. His own observations should be kept quite distinct from any information given to him by others. He may base his conclusions on the former, but not on the latter. From the common forms of law in this country, a person charged with the crime of poisoning may remain imprisoned, if at a distance from the metropolis, for some months before he is brought to trial. It is obvious, that however clear the circumstances may at the time appear to a practitioner, it will require more than ordinary powers of memory to retain, for so long a period, a distinct recollection of all the facts of a case. If he is unprovided with notes, and his memory is defective, then the case will turn in favor of the prisoner, for he will be the person to benefit by the neglect or bad memory of the witness. In adopting the plan here recommended, such a result may be easily prevented. It may be remarked that the law relative to the admissibility of notes or memoranda in evidence is very strict, and, in trials for murder, is rigorously enforced by the judges. In order to render such notes or memoranda admissible, it is indispensably necessary that they should be taken by the witness at the time the observations are made, or as soon afterwards as practicable; and, further, it must be remembered that a witness can refer to them only for the purpose of *refreshing* his memory.

Medico-legal Reports.—One of the duties of a medical jurist is to draw up a report of the results of his examination: 1, in regard to symptoms in the living; 2, in regard to appearances after death; and 3, in regard to the results of an analysis. With respect to the two first divisions of the report, I must refer the reader to the rules for investigating cases of poisoning (*ante*, pp. 93 and 137). It need hardly be observed that the time at which the person was first seen, and the circumstances under which the attendance of the practitioner was required, as well as the date of death, should be particularly stated. The hour, the day of the week, and the month, should be invariably mentioned. Some medical witnesses merely state the day of the week, without that of the month, or *vice versâ*. At a trial this sometimes creates great confusion, by rendering a reference to almanacs necessary. The words yesterday, next day, etc., should never be used. The facts which it will be necessary to enter in the report are specially stated under the heads of investigation. If these facts are not observed in the order there set down, their value as evidence of the cause of death or of the criminality or innocence of particular persons, will be entirely lost. In drawing up a report of symptoms and appearances after death, the facts should be in the first instance plainly and concisely stated *seriatim*, in language easily intelligible to non-professional persons. A reporter is not called upon to display his erudition, but to make himself understood. If technical terms are employed, their meaning should be stated in parentheses. When a subject is thoroughly understood, there can be no difficulty in rendering it in simple language; and when it is not well understood, the practitioner is not in a position to make any report. Magistrates, coroners, and barristers are very acute, and easily detect ignorance, even when it appears under the mask of erudition.

In recording facts, a reporter should not incumber his statements with opinions, inferences, or comments. The facts should be first stated, and the conclusions should be reserved until the end of the report. The language in which conclusions are expressed should be precise and clear. It must be remembered that these are intended to form a concise summary of the whole report, upon which the judgment of a magistrate, or the decision of a coroner's jury, will be ultimately based. They should be most strictly confined to the matters which are the subject of inquiry, and which have actually fallen under the observation of the witness. Thus, they commonly refer to the following questions: What was the cause of death? What are the medical circumstances which lead you to suppose that death was caused by poison? What are the circumstances which lead you to suppose that death was *not* caused by natural disease? Answers to one or all of these questions comprise, in general, all that a reporter is required to introduce into the conclusions of his report.

The reporter must remember that his conclusions are to be based only upon *medical* facts—not upon moral circumstances, unless he is specially required to express his opinion with regard to them when they are of a medico moral nature. Further, they must be founded only on what *he has himself seen or observed*. Any information derived from others should not be made the basis of an opinion in a medico-legal report. It is scarcely necessary to remark that a conclusion based upon mere *probabilities* is of no value as evidence.

In drawing up a report on the *results of a chemical analysis*, the following rules should be borne in mind. A liquid or solid may be received for analysis. 1. When, and of whom, or how received. 2. In what state was it received; secured in any way, or exposed? 3. If more than one substance received, each to be separately and distinctly labelled; appearance of the vessel, its capacity, and the quantity of liquid (by measure) or solid (by weight) contained therein. 4. Where and when did you proceed to make the analysis, and where was the substance kept during the intermediate period? 5. Did any one assist you, or did you make the analysis yourself? 6. Physical characters of the substance. 7. Processes and tests employed for determining whether it contained poison. All the steps of these processes need not be described; a general outline of the analysis will suffice. A magistrate may thus satisfy himself by an appeal to others (if necessary) whether the analysis has or has not been properly made. 8. Supposing the substance to contain poison, is this in a pure state or mixed with any other body? 9. The strength of the poison, if an acid, or if it be in solution: in *all* cases the *quantity* of poison found, determined if possible by actual weighing. 10. Supposing no poison to be contained in it, what was the nature of the substance? Did it contain anything of a noxious nature, *i. e.*, likely to injure health or destroy life? 11. Could the supposed poisonous substance exist naturally or be produced spontaneously within the body? 12. Was it present in any of the liquids or solids employed in the chemical analysis? 13. Was it contained in any of the articles of food or medicine taken by the deceased? 14. Is its presence to be ascribed to the use of any mineral matter employed by injection after death for the preservation of the body of the deceased? 15. What quantity of poison was actually separated in the free or absorbed state? 16. How much of the substance found would, under the circumstances, be likely to destroy life?

There are few reports in which answers to many of these questions, although not formally put, will not be required; and unless the whole of them are borne in mind by the operator at the time an analysis is undertaken, those which are omitted can never receive an answer, however important to the ends of justice that answer may ultimately become.

CHAPTER XIX.

THE EVIDENCE OF POISONING FROM CHEMICAL ANALYSIS—CHEMICAL EVIDENCE NOT ABSOLUTELY NECESSARY—ALL POISONS NOT DETECTABLE BY CHEMISTRY—RULES FOR CONDUCTING A MEDICO-LEGAL ANALYSIS—EVIDENCE FROM TRACES OF POISONS—DIALYSIS OF LIQUIDS—FAILURE OF CHEMICAL EVIDENCE—CAUSES—LOSS OF POISONS BY ABSORPTION AND ELIMINATION—DECOMPOSITION IN THE LIVING AND DEAD BODY.

Convictions without Chemical Evidence.—It has been supposed that chemical evidence of poisoning was always necessary, and that the *corpus delicti* was not made out unless the poison were discovered by a chemical analysis. This, however, is not a correct view of the matter. There are many poisons which cannot at present be detected by chemical analysis, and among those susceptible of analysis, there are numerous circumstances which, irrespective of a criminal tampering with the viscera, may occur to prevent their detection in the food, the vomited matters, or the contents of the stomach and bowels. (See the cases of Dr. Alexander, *ante*, p. 41; and Humphreys, *ante*, p. 85.) If such a principle were recognized by law, many criminals would escape conviction. All that is required legally, is that there should be satisfactory proof of a person having died from poison; the discovery of poison in the body is not necessarily evidence of its having caused death, nor is its non-discovery evidence that death has not been caused by it. If by symptoms and appearances, with or without moral circumstances, it can be made clear to the minds of a jury that death has been caused by poison, nothing more is required; the evidence from chemical analysis may be then safely dispensed with. Hence, it must not be supposed that a charge of poisoning cannot be sustained in the absence of chemical proof of the nature of the substance taken. The fact of a poison having been used, as well as its nature, may be determined by physiological and pathological evidence, as well as by other circumstances. In the case of Donellan, already referred to (p. 86), the only evidence of the nature of the poison used, was the odor of laurel-water perceived by the mother of the deceased. The effects which followed made up for the want of chemical proof of its nature. As some objections have been offered to the propriety of a conviction in this case, I may refer to others—one the case of a man named Thom, tried at the Aberdeen Autumn Circuit, 1821, for poisoning a person named Mitchell with arsenic. No trace of poison could be detected; but a conviction very properly took place on evidence from symptoms and appearances, coupled with moral circumstances. Another instance occurred at the Monaghan Lent Assizes, 1841, where a woman was convicted of poisoning her husband, although the nature of the poison could not be determined by the most carefully conducted chemical analysis. The poison was considered, from the alleged taste and symptoms, to have been aconite.

In the case of Humphreys (*ante*, p. 85), the medical and moral facts rendered it clear that the man had died from the effects of oil of vitriol, administered to him by his wife; but not a trace of this poison could be detected in the body of the deceased, although he survived the effects only two days. The jury, however, were satisfied, from other proofs, that death had been caused by poison, and the wife, who committed the crime, was convicted and executed. In the case of Dr. Castaing, who was tried in 1823, before a French tribunal, for the murder of his friend Ballet, by the administration of morphia, no poison was found in the body by several eminent chemists; yet the symptoms and moral evidence were considered to have furnished satisfactory proof of the crime, and the prisoner was convicted and executed. The case of the notorious William Palmer, furnishes an additional illustration, if any were needed, that medical and moral, without chemical evidence, will suffice to procure a conviction in a charge of poisoning.

It is, therefore, now a well-known and admitted fact, that a person may die from poison, and no poison be found by chemical analysis in the body. There is a popular but erroneous notion, that, if poison cannot be produced from a dead body in a visible and tangible form, then, supposing proper skill to have been employed, the only inference to be drawn is, that no poison was taken, and that death was caused by disease. This would be bringing the question of death from poison to a very simple issue indeed. It would be casting aside physiology and pathology, and requiring our law authorities to place entire and exclusive confidence in the crucible and test-tube of a chemist—sometimes prepared to swear to the presence of a quantity which he can neither see nor weigh! There are many poisons which do not admit of detection by chemical analysis, but their lethal nature may be clearly established by physiological experiments. Thus there are no chemical processes by which the poison of castor-oil seeds—of the common laburnum—of the poisonous fungi—of darnel, the Calabar bean, the poison of the œnanthe crocata, and some other vegetables—can be separated and demonstrated to exist after death, in the blood, liver, or tissues? Hence the statement that no person can die from poison, except the poison be found in the body, is a mockery, a delusion, and a snare, admirably adapted to cover a multitude of secret deaths from poison, which, but for this dogma, might be revealed by pathology and physiology. It is all the more dangerous, because the history of crime shows us that the arts of the murderer, especially of the scientific or professional murderer, are daily becoming more refined. I might add largely to the list of poisons which either by their nature, by their tremendous power in very small doses, or by the mode in which they are introduced into the system, would infallibly produce death without leaving a physical or chemical trace of their presence in the body. Even in arsenical poisoning, there may be thus an entire failure of chemical evidence. (See case by Sir R. Christison, *op. cit.*, p. 319.)

A medical jurist must, however, take care not to fall into the opposite extreme, and give an opinion that death has taken place from poison when no poison can be found, and there is no supplementary medical evidence of poisoning. The case of Mrs. Gulliver, on whose body an inquest was held at West Haddon, in Northamptonshire, in January, 1874, furnishes a sad illustration of the danger of basing a medical opinion of death from poison on hypothetical grounds. The deceased, æt. 74, died suddenly after a few days' illness. On the night before her death, a medical man saw her and found that she had disease of the heart. He gave a certificate accordingly. A rumor was circulated that she had died from poison, and a month after death her body was exhumed and examined. All the organs were found healthy excepting the heart, which was in a state of advanced fatty degeneration.

A chemical expert, Mr. Rodgers, made an analysis of the viscera, and stated that he could find no poison which would account for death; but he had detected "traces" of morphia. (On this alleged finding of traces, some remarks have been elsewhere made. See *ante*, p. 144.) "With the state of heart described, he should have expected Mrs. Gulliver to have had fainting attacks, and at any time a *fatal syncope.*" In spite of the opinion thus given, and which was thoroughly consistent with the facts proved, he went out of his way to suggest that the cause of death was "some volatile noxious substance given to the deceased immediately prior to death, but which he was unable to detect."

The usual medical attendant of deceased had in his evidence stated that when he saw the body, soon after death, it had a high temperature, *i. e.*, that it was unusually warm. On this loose observation, the chemical expert based his opinion that Mrs. Gulliver had died from some volatile poison, and not from disease. The only person with her at the time of death was a niece, the wife of a medical practitioner, who was acting as nurse to her aunt. On hearing of the charge of murder, thus brought against her by implication, she destroyed herself with poison.

It is scarcely necessary to say that the medical opinion was based on a pure mistake, and on an entire ignorance of the conditions required for the cooling of the body. The disease of the heart was alone sufficient to account for sudden death in an aged woman. There were no symptoms or appearances indicative of poisoning; there was no poison in the body to account for death, and the suggestion that a volatile poison had been used just before death, which could not be detected by chemical processes, can only be described as a reckless hypothesis, without any reasonable foundation. If loose statements of this kind are received as medical evidence in the absence of facts, any person who is in attendance on the dying, may be involved in a charge of murder! (*Med. Times and Gazette*, Jan. 17th, 1874, p. 72. *British Med. Jour.*, Jan. 17th and 24th, 1874, pp. 89, 113, 125. *Pharm. Jour.*, Jan. 17th, 1874, p. 584, and *Guy's Hosp. Reports*, April, 1874.)

When the other branches of evidence (symptoms and appearances) are weak or defective, the detection of poison by chemical analysis is of such importance, that if it fail, an acquittal will follow. Conjoined with strong moral circumstances, chemical evidence will often lead to conviction when the appearances in the body are entirely wanting, and the evidence from symptoms is imperfect. The great value of chemical evidence, in otherwise doubtful cases of poisoning, is frequently shown in the detection of poison in bodies which are exhumed many months or years after burial, when all appearances are destroyed by decomposition. We cannot therefore be surprised to find that it is this branch of evidence which is deemed most satisfactory to the public mind, and which is earnestly sought for by our law authorities on charges of poisoning. The reason is that, in most cases, in the hands of a trustworthy and discreet analyst, it demonstrates at once the means of death; while symptoms and appearances, especially when deposed to by inexperienced witnesses, are fallible criteria.

Rules for conducting an Analysis.—Before proceeding to the analysis of any suspected substance, we should, if possible, make ourselves fully acquainted either with the symptoms or appearances, or both, observed in the person suspected to have been poisoned. We may by a knowledge of these facts determine, *a priori*, whether we shall have to search for a neurotic, irritant, or corrosive substance. The kind of poison may often be predicted from the symptoms and appearances, and our analysis directed accordingly.

The chemical evidence may be divided into several branches. The analysis may extend—

1. To the pure poison. We may be required to state the nature of a substance (part of the poison administered) found in the possession of a prisoner, or lying near the deceased.

2. The analysis may be confined to a portion of the substance of which the affected party partook; and here the poison is usually mixed up with liquids or solids of an organic nature. The steps of the analysis are then rather more difficult. *a.* There may have been various substances combined in a meal, and the poison have been mixed with one substance only. This will show the necessity for examining separately the various articles used at a meal, if we wish to discover the real vehicle of the poison. *b.* Symptoms of poisoning may occur after the eating of a pudding. A part of the pudding may be analyzed, and no poison discovered; because the poison, instead of being incorpo-

rated with the dough, may have been loosely sprinkled like flour over the exterior only. *c.* A similar circumstance may occur in the poisoning of a dish of meat. The gravy may be poisoned, and not the meat. A case of this kind occurred to Sir R. Christison. A whole family was attacked with symptoms of poisoning after a meal on roast-beef. The meat was examined, but no poison could be discovered. It was then ascertained that the poison had been mixed with the gravy, and that those who had taken the meat without the gravy, had suffered but slightly. In one instance, referred to me, arsenic was placed instead of salt on the edge of the plate of the deceased (p. 88; *The Queen* v. *Jennings*, Berks Lent Assizes, 1845). No other person experienced symptoms of poisoning after the meal, except the child who ate out of that plate. In the case of Bodle, tried in 1833, the deceased was proved to have been poisoned by arsenic administered in coffee. The coffee was kept ground in a bottle, to which every one of the family had access; and there could be but little doubt, from the circumstantial evidence, that the poison had been mixed with the coffee in this bottle. That which remained in the bottle was carefully examined by the late Mr. Marsh, but no trace of arsenic could be detected. The poison had most probably been mixed with the *upper stratum* only of the powdered coffee, and the whole of the poisoned portion had been used for breakfast. A remarkable fact was brought out in the case of the *Queen* v. *Edwards* (Central Criminal Court, November, 1844). The deceased, it was stated, had died from drinking part of the contents of a bottle suspected to contain sugar of lead—but it was proved that some of the same liquid had been drunk by another person the night previously without any injury resulting. The medical witness explained this by saying that the poison existed as a crust in the bottle, which might have been detached in one case and adherent in the other. A somewhat similar case is given under the section on CARBONATE OF LEAD. (See that compound, *post.*) Facts of this kind are of some medicolegal importance; they will often enable a witness to explain certain anomalies in cases of poisoning. By bearing them in mind, it is easy to understand how it is that one or two persons only will suffer at a meal made in common or on the same article of food, while others will escape (p. 88).

3. The chemical analysis may be directed to the matters *vomited* and evacuated. In irritant poisoning, a large quantity of poison is often expelled in this manner, and may be detected especially in the matter *first* vomited. In a suspected case, an immediate analysis should be made of the matters ejected from the stomach. They may be regarded as furnishing to the medical jurist the proofs required to establish the *corpus delicti*. Supposing that the results of the analysis of vomited matters are negative, the urine or an evacuation should be examined. Within two or three hours after the suspected substance has been taken, if mineral poison, it may be usually detected in the urine.

4. If death has ensued, an analysis of the contents of the stomach and intestines must be made. Supposing no vomiting to have occurred, or that this has been slight, and death has taken place speedily, then we may expect to find abundant evidence of the poison in the viscera.

If no poison should be found in the stomach, the contents of the duodenum and the other small intestines, as well as of the rectum, must be separately examined. If the poison cannot be detected in the contents of the stomach and intestines, it must be sought for in the tissues of the viscera, especially of the liver and spleen.

As a summary of the various conditions under which poison may be discovered by chemical analysis, it may be stated, that in the *living* body, chemical evidence is derivable from an examination: 1, of the matters vomited; 2, of the evacuation; and 3, of the urine; in the *dead* body poison may be found *free.* 1, in the stomach; 2, in the small intestines; 3, in the rectum;—or *absorbed:* 4, in the blood; 5, in the liver; 6, in the spleen and kidneys; 7, in the heart; 8, in the lungs; 9, in the muscles.

It is obvious that one or several of these sources of chemical evidence may be wanting, and it is rare in any one case of criminal poisoning that all are open to a medical witness. The detection of poison in the vomited matters during life, and in the stomach, intestines, liver, or other organs after death, is, of course, the most satisfactory kind of chemical evidence; since, *cæteris paribus*, it is a clear proof of poison having really been taken. It is difficult to admit the supposition that it should have been designedly introduced after death; besides, in this case, the absence of all marks of vital reaction, and of any symptoms during life indicative of poisoning, would remove such a suspicion, should it arise. The detection of poison in the urine actually passed by the patient, is a clear proof that it has been absorbed and eliminated from the living body. If the poison be detected as a deposit in the tissues of any of the organs, and due allowance be made for imbibition from the adjacent viscera, there can be no doubt of its having been introduced into the body during life. The presence of poison in the stomach and bowels, or their contents, with such marks of vital reaction as are known to be produced by the particular substance, as, for instance, inflammation in the case of the irritants, affords the strongest presumptive evidence of death from poison, open to be rebutted by other proofs of death from disease, under which the deceased may have been laboring at the time.

Medical Evidence from Traces of Poison.—"Traces," or unweighable quantities, are the *ignes fatui* of toxicologists. One analyst will rely upon the chemical effects produced by the five hundredth part of a grain, another will put his faith in the thousandth, and a third will even go to the one hundred-thousandth or the millionth! I have known an expert to arrive at a conclusion one day that he had discovered "traces" of poison, while the next day, on further consideration, he rejected the chemical results as unsatisfactory, simply because the quantity was too small to admit of any fair or reasonable corroboration. In an examination, made some years since, of an important case of alleged murder by poisoning, in which I was joined with the late Dr. Miller and another experienced chemist, two of us considered that there were "traces" of the poison in a certain organ, and one, that they were not reliable. As there was this difference of opinion, we rejected the chemical results as unsatisfactory.

A reliance upon minute and uncorroborated results, led Orfila to affirm, erroneously as it has since been proved, that arsenic was a normal constituent of the animal body. An undue confidence in the tests for morphia, when applied to organic liquids, led an English "expert" to swear to the presence of traces of this alkaloid in a stomach, and to its having been the cause of death, in a case in which he was subsequently obliged to admit that he was in error. (*Pharmaceutical Journal*, Jan. 1855, p. 350; and Feb. 1858, p. 443; also *Guy's Hospital Reports*, Oct. 1857, p. 497.) In a more recent case another expert of less experience swore positively to the presence of "traces" of morphia in the contents of a stomach of a lady when there was no evidence to show that any had been administered to her or that she had suffered from any of the symptoms of poisoning with morphia. (Case of Mrs. Gulliver, Jan. 1874, *ante*, p. 149.)

Beyond doubt, some poisons may be detected in smaller quantities than others. A witness might rely upon traces of arsenic, but he would hesitate to swear to the presence of such an alkaloid as morphia when operating on an unweighable quantity. No toxicologist has worked this question to a greater or more minute extent than Dr. Wormley, an American professor of chemistry and toxicology. He has determined the limits of all the known tests for all ordinary poisons. (*Micro-Chemistry of Poisons.*) Referring to morphia, he says (p. 483): "No one of the tests, taken alone, is peculiar to this alkaloid. . . . It must be admitted, however, that in regard to delicacy of reaction the tests at present known for the identification of morphia are inferior to those of many of the other alkaloids, and moreover, this alkaloid is more difficult than most others to separate from foreign organic substances."

Leaving out of the question the skill of the analyst and the nature of the substance, there is one universal and satisfactory test by which the existence of "traces" of poison may be confirmed or disproved. No poison can be taken into the living body or remain there without producing, sooner or later, certain well-known effects. If this physiological evidence is not forthcoming, it is far more probable that the "unweighable" substance has deceived the analyst, than that an active poison can have been taken without producing symptoms or destroying life. Cases of this description, affecting as they do life and reputation, show the absolute necessity of a public prosecutor competent to sift evidence before the exhumation of a dead body is allowed to take place.

When but infinitesimal traces of poison are discovered, and large quantities of chemicals have been used for its extraction, as in what Sir R. Christison has properly designated the "enthusiastic" analysis of some French medical jurists, it would be unsafe to base any conclusion upon the results. Thus, in a *cause celèbre* which occurred in 1840, in France (case of Madame Laffarge), the medical witnesses, in order to extract the poison, boiled up the body with many gallons of water and acids. The body of the husband underwent decoction in large iron cauldrons outside the court, while the wife was on her trial for the murder within! The quantity of sulphuric acid, nitric acid, and nitre, which must have been used on this occasion was so great, that there was good reason to suspect the probable introduction of small traces of poison *ab extra*—the whole quantity of arsenic found in the body not exceeding the one hundred and thirtieth of a grain. A jury would, undoubtedly, be fully justified in rejecting chemical evidence procured by such means.

Causes of the Non-Detection of Poison.—But let us take the case, that chemical evidence is entirely wanting, and that no poison is detected under any of the circumstances mentioned: if there be other facts to render death from poisoning probable, we must endeavor to explain why this important branch of evidence has failed. There are few medical jurists who have not met with cases in which, although undoubtedly death was occasioned by poison, either irritant or narcotic, not a trace of the noxious substance could be detected in the solids or liquids of the body. The non-discovery of poisons in cases of poisoning may depend:

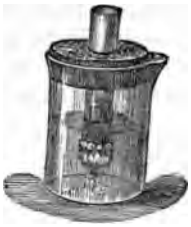
1. *On the Nature of the Poison.*—Mineral poisons, if present, may in general be identified with certainty in the dead body, but the greater number of vegetable poisons, when diffused through the body, are beyond the reach of chemical analysis. Botanical characters may sometimes serve to point out the nature of the substance; but only in those instances in which the plant has been swallowed in the state of leaves, roots, or seeds. If the extract or inspissated juice has been administered, or if the poison were in the form of infusion, tincture, or decoction, a chemical analysis will commonly be of no avail. The processes for the separation of the alkaloids, morphia, strychnia, brucia, veratria, nicotina, and others, have been greatly improved, but they still involve many difficulties compared with mineral poisons. Persons who are killed by these powerful agents commonly die rapidly, so that there is no time for the deposit of any large amount of poison in the liver or other organs. Some of the alkaloids, when isolated from organic matter in a pure state, admit of detection in any quantity visible to the eye, up to the thousandth of a grain or less, but the practical difficulty is to separate them from the large amount of organic matter in which they are contained, in a state sufficiently pure for the application of tests.

Dialysis.—It would be impossible to apply chemical tests to the liquids of the stomach and intestines; but the process of dialysis, as suggested by the late Professor Graham, will often enable a medical jurist to procure rapidly a portion of liquid sufficiently free from organic matter for preliminary testing. This process can only be applied to substances dissolved. All crystallizable or crystalloidal bodies soluble in water, and all the acids can be thus separated from blood, mucus, milk, gruel, and other viscid articles of food.

A thin layer of gutskin or parchment is tied securely round the open mouth of a short wide test-tube, which has been cut so as to be open at the other end. This is fitted to a hole in a layer of cork, and the cork is fitted to a beaker, so that the tube may be raised or lowered at pleasure. A small portion of the contents of the stomach, if very viscid, should be diluted with a little water, and if not acid, acidulated with acetic acid. A small quantity of distilled water is now placed in the beaker, and the tube containing the liquids for dialysis is lowered into the water, and it is allowed to remain there for two or three hours.

Some indications of the nature of the poison may then be detected in the water by the ordinary tests; but the process will not be complete until after the lapse of twenty-four hours. The dissolved poison is slowly transferred to the water through the membrane, while the colloid organic substance remains in the tube. The quantity of distilled water placed in the beaker should be about four or five times as much as that of the organic liquid in the tube. It may be easily concentrated by evaporation, and one portion tested for the metals by sulphuretted hydrogen, a second for the acid poisons, and a third for the alkaloids.

FIG. 3.

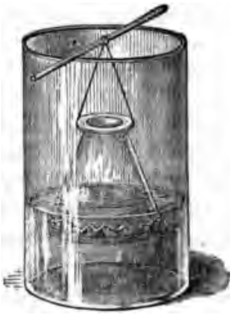


Beaker and tube for the dialysis of liquid poisons.

For this separation of the alkaloids, a larger proportion of material is required, and a glass-vessel of a conical shape is convenient. The level of the liquids should be the same in the beaker and dialyzer, and gutskin, or thin bladder without holes will be found better than parchment paper.

This process is chiefly fitted for the preliminary testing of the fluid contents of the stomach. It is impossible by it to separate the whole of the poison, and it frequently happens that a small quantity of organic matter, including some coloring matter, passes through the membrane with the poison.

FIG. 4.



Dialyzer for alkaloids.

Poisons that are of a highly *volatile* nature may be speedily dissipated; so that in a few hours, or a few days after death, none may be discovered. Alcohol is well known to pass away so rapidly, that no spirituous odor may be perceived in the contents of the stomach, although the person may have died speedily, and the body be inspected within six or eight hours after death. Prussic acid, chloroform, and nicotina may be in like manner rapidly lost or decomposed. (See PRUSSIC ACID.)

2. *Influence of Vomiting and Purging.*—The non-detection of poison in the stomach and bowels may be owing to its having been expelled by excessive *vomiting and purging.* In all such cases, however, the poisonous substance ought to be found in the vomited matters, if these are forthcoming. Vomiting and purging cannot, however, affect that portion of poison which has been absorbed and deposited in the organs. The quantity actually taken by deceased has not so much influence on the power of detection as the quantity which remains in the body at the time of death. This is subject to great variation, according to the time which the person survives.

3. *Loss by Absorption and Elimination.*—Solid mineral poisons are

usually detected without difficulty, because they are generally administered criminally in large doses; but in cases of chronic poisoning, *i. e.*, where the substance is administered in small doses at long intervals, chemical analysis will sometimes fail; for the poison may have become entirely absorbed and eliminated. Thus, if the dose of arsenic be small, if the poison be taken in a state favorable for absorption, *i. e.*, in solution, or the person survive for a period of two or three weeks, no trace of the substance may be found in the body. (See *ante*, *Elimination of Poisons*, p. 42.)

A remarkable case of chronic poisoning with arsenic and the entire failure of medical evidence to show its presence in the body is related by Sir R. Christison to have occurred in Scotland. (*Op. cit.*, p. 319.)

When the poison has been administered in very small quantity none may be found after death. Dr. Edwards has reported the cases of five children poisoned by colored sweetmeats; the poison had been taken in small quantity and in repeated doses. Three died, but arsenic was found only in the stomach of one of these, although the cause of death was the same in all. (*Pharm. Journal*, February, 1857, p. 417.)

4. *Decomposition of Poison in the Living Body.*—It has been long known to toxicologists that although phosphorus, by reason of the luminosity of its vapor in the dark, is susceptible of detection in the most minute quantities, it is rare that any of the poison can be detected in a case in which the person has survived a few hours or days. This is owing to its rapid oxidation in the living and dead body, when the phosphorus is in a finely divided state. It is thus converted into liquid phosphoric acid, which disappears with the fluids so as to be no longer traceable with chemical tests. (See a paper on this subject by Dr. Lefort, *Ann. d'Hyg.*, 1874, vol. 1, p. 405.) It has been frequently a subject of remark that in poisoning with opium, if the person survive many hours, no trace of the substance, either as opium, morphia, or meconic acid, has been found in the stomach, bowels, or tissues. Several cases of this kind have occurred to Sir R. Christison (*On Poisons*, 4th edition, p. 697); and others of a similar nature have occurred to myself. In a case of death from arsenic in April, 1858, I had an opportunity of again examining this question. The deceased, under medical treatment, took, twelve hours before death, five grains of calomel with one grain and a half of opium, and again four hours before death two grains of calomel and one and a half grain of opium, making three grains of opium and seven grains of calomel. Mercury (derived from the calomel) was found in large quantity in the stomach and duodenum, but there was no trace of meconic acid or morphia (opium). There was no mercury in the lower bowels. Arsenic was found with the mercury.

In August, 1857, I was consulted respecting a case of alleged poisoning with opium, which was the subject of a trial for murder at the Liverpool assizes. A child died under the usual symptoms of narcotic poisoning, and it was proved that the mother, who was charged with the murder, had procured from a druggist, on the day before the death, one hundred drops of laudanum. On inspection, there was congestion of the brain and its membranes, but no natural cause of death. Mr.

Stone, of Manchester, made an analysis of the stomach and viscera. No morphia—the poisonous alkaloid of opium—was found anywhere; but from one result Mr. Stone was led to suspect the presence of meconic acid. He declined, however, to speak positively to this, or to state that the result unequivocally proved the presence of opium in the stomach. There could be no doubt, from the medical evidence, that death was caused by opium, but no morphia could be detected in the body, although the child could not have survived many hours. Some remarks have been elsewhere made on the changes which poisons may undergo after their absorption. (See *ante*, p. 62.)

5. *Decomposition in the Dead Body.*—Putrefaction does not materially affect mineral poisons. Antimony and arsenic are liable to be converted into colored sulphides as a result of the production of sulphuretted hydrogen. In this state, the sulphides become permanently fixed in the solids, dyeing them orange or yellow. A metallic poison may be changed in its chemical composition but it is not lost. The metal may be recovered from any portion of the decomposed remains.

Volatile poisons are lost, with the exception of prussic acid, which, under the production of sulphide of ammonium during putrefaction, is converted into sulphocyanide of ammonium, a fixed but soluble salt.

Nicotina and conia, being volatile alkaloids, may disappear during putrefaction by oxidation or chemical changes. Strychnia appears to withstand the process. It has been discovered many months after death in highly putrefied organs. With regard to morphia, brucia, and veratria, reliable facts are wanting to show how far they are changed as a result of decomposition in the dead body.

6. *Influence of the Quantity Taken.*—The power of detecting poison in the dead body must depend, not on the dose taken, but on the quantity remaining in the stomach and other organs at the time of death. However large the dose, if the person has survived some hours or days, the residual quantity may be very small. Again, the dose originally taken may have been so small, that by the mere effect of diffusion, it would be difficult, if not impossible, to trace it. I have elsewhere published the case of a child that died in four hours, from the sixteenth part of a grain of strychnia, and that of an adult who died in twenty minutes, from half a grain. (*Guy's Hospital Reports*, Oct. 1856, p. 138.) Such small quantities, absorbed and diffused by the blood through the whole of the body, would, as a general rule, be beyond the reach of a chemical analysis which would be satisfactory on a charge of murder. Assuming in the case of the adult, that the half grain was entirely absorbed, and that it remained unchanged in the blood, the proportion of strychnia in a pound of that blood would not exceed the fiftieth part of a grain, or one eight-hundredth of a grain in an ounce! In the case of the child, if none were eliminated or unchanged in the body, the proportion would be only the one two-hundred-and-fortieth of a grain in a pound of blood, or about one four-thousandth of a grain in an ounce! It is not probable that such minute quantities could be detected.

A similar remark may be made with regard to children poisoned by opium. In November, 1874, three children died in a few hours with all the usual symptoms of narcotic poisoning after a teething powder had been given to each. Each powder contained about one-tenth of a grain of opium, equal to the one-hundredth of a grain of morphia. The greater part, if not all, of this quantity would be absorbed and diffused over the body in a few hours. It appeared to excite some surprise that no morphia could be detected in the stomachs; but the symptoms proved that the alkaloid had been absorbed.

CHAPTER XX.

OBJECTS OF A CHEMICAL ANALYSIS—NATURE OF THE POISONS—INFERENCES FROM THE QUANTITY FOUND IN THE BODY—PROOFS OF ADMINISTRATION FROM CHEMICAL ANALYSIS—CASES—DANGER OF PREMATURE OPINIONS.

Objects of a Chemical Analysis.—A chemical analysis is commonly directed in toxicology to the determination of the following points: 1. The *nature* of the poison. 2. The proportion, or *quantity*, in which it has been taken. 3. The solution of certain questions connected with the criminal administration of poison.

1. The *nature* of the poison and the probable quantity administered, are usually stated in an indictment; but it is not absolutely necessary for conviction, that the substance thus stated, should be proved to have been that which was actually administered. There were some medical difficulties formerly connected with this subject, since, on an indictment for poisoning, it was always necessary to prove that death was caused by poison; but the person may be indicted for murder, and the proof of the means of death is not now indispensable. By the statutes 14 and 15 Vic., cap. 100, s. 4, it shall not be necessary "to set forth the manner in which, or the means by which the death of the deceased was caused; but it shall be sufficient in every indictment for murder, to charge, that the defendant did feloniously, wilfully, and of his malice aforethought, kill and murder the deceased." It must, however, be shown that the substance or substances administered, were of a noxious or poisonous nature, and either caused or accelerated death.

2. The *quantity* of poison administered is generally stated conjecturally; but when any portion of the original vehicle of the poison is discovered, it is in the power of a witness to give a tolerably accurate opinion of the quantity taken. Thus, all solid substances given for analysis should be first weighed, and all liquids measured—a quantitative analysis may then be performed at any subsequent period. The chief question in law in regard to the quantity of poison is: whether it was sufficient to destroy life, or to produce any noxious effects? The malicious intention of a prisoner may be sometimes inferred from the quantity of poison existing in the substance administered.

It need hardly be observed, that the *quantity remaining in the stomach*, or the portion of *absorbed* poison deposited in the tissues, can give no idea of the quantity actually taken by or administered to the deceased; since more or less of it may have been removed by vomiting and purging as well as by elimination. But the quantity found free in the stomach and bowels, even after a portion has been thus lost, is often more than sufficient to destroy the life of a human being. It is singular that, notwithstanding the existence of these very obvious and natural causes for the removal of poison from the stomach, barristers should so frequently address the inquiry to a medical witness—whether the quantity which they found in the body was sufficient to cause death? Whether this qestion be answered in the affirmative or negative, is a matter which, medically speaking, cannot at all affect the case, since either no traces of poison, or but a very small portion, may be found in the viscera, and yet the deceased may have assuredly died from its effects. Absorbed arsenic, as it exists in the tissues, is never found except in minute proportion, a proportion commonly insufficient to destroy the life of another. Hence, whether much or little is detected, the question is misleading; since the fact of death having been caused by poison does not, in the least degree, depend upon the precise quantity which happens to remain in a dead body. It has been truly remarked by Orfila, in regard to arsenic, and it equally applies to all poisons, that that portion which is found in the stomach *is not that which has caused death;* but the *surplus* of the quantity which has already produced its fatal effects by its absorption into the system. The inquiry should therefore be directed to the probable quantity of poison *taken;* not to how much remains in the body.

This question is one of more importance than may at first sight appear. There is scarcely a trial for criminal poisoning, in which it is not put to a medical witness, either by the judge, or the counsel for the prosecution or defence. Supposing poison to be found in the stomach, but not in sufficient quantity to destroy life—is it therefore to be assumed that the person did not die from its effects? This would be equal to laying down the doctrine, in face of the most indisputable evidence to the contrary—that poisons, when taken into the body, are not liable to be expelled by vomiting or purging, or to be removed from the stomach by absorption and carried out of the body by elimination. The real object of a toxicologist is to discover the poison by clear and undoubted evidence. If more than sufficient to cause death be found in a dead body, then the dose must have been larger than was necessary; but if this proof be always required, what is to become of those cases of criminal poisoning in which the prisoner administers a dose only just sufficient to destroy life, or in which the deceased, by the strength of his constitution, happens to survive the effects for some days or weeks, and ultimately dies of exhaustion? No poison would be detected under these circumstances. (See the case of Dr. Alexander, *ante*, p. 41.) Orfila has most completely demonstrated the fallacy of this objection to medical evidence, and the danger of a court of law relying upon it. (See *Ann. d'Hyg.*, 1845, vol. 1, p. 347; also *Toxicologie*, vol. 2, p. 731.)

When the *quantity* of poison found in a dead body, either in the free or absorbed state, is *small*, it is of course a fair question whether it may not be the remains of doses given medicinally, and without criminal intention. Arsenic and antimony are frequently used in medicine; and if a person dies while taking Fowler's mineral solution, or solution of tartar emetic, one or both of these metals may be found in the stomach or deposited in the liver. I am informed that in the fen districts, Fowler's solution of arsenic is much used by the poor as a preventive of ague; and that they readily procure it from druggists for domestic use without a medical prescription. Accidents sometimes occur to children by reason of an overdose, given with innocent intention. Small quantities of mercury are not unfrequently found in examining the livers of persons who have died after medical treatment. The taking of a dose of calomel, or blue pill, shortly before death, may account for this. In the incineration of a liver, a small quantity of copper or lead may be discovered—introduced accidentally during life. Opium, morphia, strychnia, or prussic acid, may be thus found in the body of a person who has died suddenly while taking any one of these poisons medicinally. We are bound, therefore, to consider before we place any reliance on such chemical evidence as "minute," "distinct," or "unequivocal traces" of poison, whether medicinal use or accidental introduction may not account for the discovery. Who prescribed the medicine? For what was it prescribed? What were the symptoms preceding death? When the case is really of that innocent complexion which some barristers are inclined to assign to it, from the small quantity of poison found, there will be no difficulty in obtaining answers to these questions. In Wooler's case the medical men did not prescribe arsenic, and the deceased, at least some time before she died, could not have had access to arsenic. In Cook's case the medical men, called in by the criminal, did not prescribe antimony, and would not have prescribed it medicinally under the circumstances. Deceased had no access to antimony, and there was no reason for his taking it secretly. He was sick when the murderer was about him, and the sickness abated in his absence. The presence of even a small quantity of antimony in his dead body, therefore, at once explained *that* for which no other theory could satisfactorily account. The value of chemical evidence does not depend on the discovery of any particular *quantity* of poison in the stomach—it is merely necessary that the evidence of its presence should be clear, distinct, satisfactory, and conclusive. At the same time, a reasonable objection may be taken to a dogmatic reliance upon the alleged discovery in a dead body of minute fractional portions of a grain; especially in a case in which the symptoms and appearances are doubtful.

We may now take the converse proposition—*a large quantity* of poison may be found in a dead body, both in the *free* and *absorbed* state. What then is the inference? Under these circumstances, no theory of medical treatment, or of the innocent use of medicine, would be applicable. In a case of this kind, the cause of death may admit of no dispute; not because the residuary quantity of poison is large, but because the symptoms under which the person suffered, and the appearances, may have been such that there could be no room for a reasonable doubt. It is true that a large quantity of poison may be injected into the stomach or rectum after death. Such a state of things would be indicated by the absence of symptoms and appearances. These contingencies tend to show how important it is that we rely not too strongly or implicitly on mere chemical results. The discovery of *absorbed* poison removes any difficulty in respect to injection after death, and proves that the poison must have entered the body during life, provided we have satisfied ourselves that there could have been no cadaveric imbibition from the soil.

The quantity of residuary poison found in a dead body has been supposed to throw a light upon another important branch of medico-legal inquiry, namely, whether the act of poisoning was one of *murder* or *suicide*. A large quantity is considered to indicate a conscious and deliberate swallowing of the poison, and under certain limitations this is no doubt correct. It has been supposed that the *quantity* of arsenic found in the stomach and bowels may throw a light on the question, whether the poison had been taken voluntarily with the intention of committing suicide, or whether it had been criminally administered by another; *i. e.*, supposing the evidence to establish that there could have been only one act of administration. There is no doubt that a much larger dose may be taken by a suicide than could be secretly administered by a murderer; and thus, if a large quantity is found in the stomach, it is supposed to furnish a presumption in favor of suicide and against murder. Suicides have been known to take as much as two tablespoonfuls, or *one thousand grains*, of arsenic. In a case of suicide by arsenic, which occurred at the Bristol Infirmary in July, 1872, a larger quantity had been taken. Dr. Smith found, on a post-mortem examination, four ounces of arsenic in the stomach, of which two and a half ounces were in one mass. The woman, when brought to the hospital, was in a state of complete collapse. Death took place rapidly. (*Pharm. Jour.*, July 27, 1872, p. 75.)

When the poison is either in a liquid or solid form, and when it has a very strong taste and odor, it is difficult to come to the conclusion that it could have been taken unknowingly. In the case of Mr. Sadleir there was reason to believe, from the large quantity of essential oil of bitter almonds found in his body, that the deceased must have swallowed many ounces. A large dose of this oil could not be taken unknowingly, or by any accident. In the section on ARSENIC will be found a case in which a man, æt. twenty-two years, swallowed from four to six ounces of this poison. He died in four hours, having suffered from vomiting and purging. After death, *two ounces* of arsenic were found in his stomach. A liquid poison may be poured down the throat while the person is sleeping, as in the case of Humphreys (*ante*, p. 84), or it may be forcibly introduced into the mouth, as in the case of Fougnies, for whose murder, by nicotina, the Count Bocarmé was executed in Belgium; but these violent modes of administration invariably lead to detection.

The question arising from the discovery of a large quantity of arsenic in the stomach, was first seriously raised in the case of Madeline Smith (Edinburgh Court of Justiciary, July, 1857, *ante*, p. 69). The deceased, L'Angeliér, died from the effects of arsenic, under the usual symptoms of vomiting and purging, in from ten to twelve hours after, it is believed, the poison had entered his body. Although, from an absence of suspicion at the time of the illness, the vomited matters were not examined, there can be no doubt that some arsenic must have been ejected with them. On an inspection of the body, the late Dr. Penny found *eighty-eight grains* (half a teaspoonful) of arsenic in the stomach and its contents, irrespective of a portion contained in the intestines, and the quantity deposited in the viscera (*Report of Trial*, p. 51). Sir R. Christison stated, in cross-examination, that he did not recollect any case of a person murdered by arsenic in whose stomach so much as eighty-eight grains had been found after death. At the same time he admitted that a large quantity might be administered in certain thick articles of food, *e. g.*, cocoa; and that a much larger quantity must have been swallowed by deceased than was found in his stomach. This was made a turning-point of the defence; it was contended that so large a dose could not have been taken unknowingly, and, therefore, that it indicated suicide. The learned counsel argued: "It is a dose which, so far as experience goes, never was successfully administered by a murderer. There is not a case on record in which it has ever been shown that a person administering poison to another, succeeded in persuading him to swallow such a quantity." It need hardly be remarked that persuasion has nothing to do with this inquiry. Could a man unknowingly take so large a dose, if secretly administered by another? Generally speaking, persons are destroyed by a much smaller quantity than was here found; but Sir R. Christison, since the trial, has placed on record a case of *murder* by arsenic, in which from *ninety* to *one hundred grains* of this poison were found in the stomach after death! The person had survived from five to seven hours, and there had been frequent vomiting of a yellowish or greenish liquid during this period. The arsenic was administered in whisky punch with sugar, and it was kept in suspension by constant stirring (*Edinburgh Monthly Medical Journal*, December, 1857, p. 481, and *Pharmaceutical Journal*, January, 1858, p. 382).

In *Regina* v. *Dodds*, tried at the Lincoln Assizes in December, 1860, the prisoner was charged with administering arsenic to deceased with intent to murder. The quantity stated to have been found in the stomach by the medical witnesses was 150 grains. There was no reason to suppose that the man had taken the poison with suicidal intention; but on the contrary, there was strong evidence to presume that it had been administered to him with a design to destroy life. The late Mr. Justice Willes communicated to me a similar case, which was tried before him (*Reg.* v. *Hewitt or Holt*) at the Chester Winter Assizes, 1863. The prisoner was convicted of poisoning her mother with arsenic in the month of March preceding. Although the symptoms of irritant poisoning were very clearly marked, a medical man who attended her, certified the cause of death as gastro-enteritis! Eleven weeks after the burial of the deceased, the body was exhumed and examined. It was proved that shortly before her mother's death, the prisoner had purchased a quarter of a pound of arsenic for threepence, and there was clear evidence of administration, a large dose having been given to the deceased in liquid shortly before her death. The inspection revealed the extraordinary fact, that 154 grains of solid arsenic were found in the stomach alone. It had been partially converted into sulphide as a result of putrefactive changes, and it was observed that the liver, omentum, and right side of the heart were thickly stained with yellow sulphide of arsenic. Had these cases been brought forward at the trial of M. Smith, the facts would have neutralized a part of the defence on which the public were induced to place a great, but wholly unjustifiable reliance. No medical jurist could admit that the discovery of eighty-eight grains (only half a teaspoonful) of arsenic in the stomach, was inconsistent with an act of homicidal administration; and yet the defence rested in a great degree on this very slender point! In this case, no account was taken of the probability that the man was intoxicated—a condition in which a large quantity of poison might be easily administered.

3. The *administration* of poison. A careful analysis may occasionally throw light on the question—who administered the poison? In the case of Humphreys (*ante*, p. 84), the discovery of sulphuric acid on the night-dress of the prisoner was an important fact to identify the woman as the person who had administered the poison. In the case of Wooler (*ante*, p. 83), arsenic was found in a syringe used for the purpose of injection. This proved not only how the poison had been administered to the deceased, but it had a tendency to fix the crime on a particular person. In Hartley's case (Central Criminal Court, May, 1850), the prisoner, a girl, was charged with attempting to administer oil of vitriol in coffee to her father. The prisoner usually made the coffee for breakfast, and would then have had an opportunity of adding it to the liquid. The acid might, however, have been mixed with the coffee in the cup after it had been poured out; and, in this case, other persons would have had the opportunity of poisoning the coffee. This question was solved by the aid of chemistry. I procured the coffee-pot, and found that it was old and rusty; the poisoned coffee was tested, and it contained no trace of iron, but on warming a small quantity of the acid coffee in the pot, it was immediately and strongly impregnated with sulphate of iron. It was therefore clear that the acid had not been mixed with the coffee in the pot, and that it might have been afterwards put into the cup without the knowledge of the prisoner. Numerous other cases will suggest themselves in which a guilty person may be detected, and an innocent person protected, by the aid of a chemical analysis of the poisoned food.

In the case of *Reg.* v. *North* (Guildford Summer Assizes, 1846), the proof of administration rested in part on the carbonizing action of oil of vitriol on sugar. Mary North was tried for the murder of an infant by giving to it oil of vitriol. The deceased had died from the effects of the poison; therefore the only part of the case which created difficulty was the proof of administration. The mother of the deceased, wishing to give the child some aniseed-spirit and water, placed a lump of sugar in a white cup, and added a teaspoonful of the spirit; she then went to another apartment, and poured from a kettle about a teaspoonful of water. She observed no particular appearance in the mixture; she *tasted* it; there was no hot or acid taste, there was no blackening or change of color; she then gave the mixture to the infant, while a little girl who was present *drank up the dregs*, and suffered no ill effects. The prisoner was present, and in about half a minute took the child. After the child had taken this liquid, there was no symptoms or effects to attract attention, and it appeared relieved of the wind from which it had suffered. The mother left the room, and the prisoner then took the infant into an adjoining pantry, in which it was sworn there was a bottle of vitriol, put there by the prisoner. In about a minute and a half or two minutes, the mother, owing to a noise which she heard, returned to the room, and found her infant evidently writhing in great pain, and its mouth covered with a whitish froth. The prisoner, while bringing the child from the pantry into the kitchen, was, according to one witness, in the act of wiping its mouth with a napkin, on which sulphuric acid was found. Medical assistance was immediately sent for; but, in spite of the best treatment, the child died. The defence was, that the mother had made a mistake, and given a teaspoonful of oil of vitriol in place of aniseed; but that was impossible under the circumstances, as the child who drank the dregs of the mixture did not suffer, and the mixture could not have been made as described by the mother, without blackening the sugar. In spite of these obvious conclusions, the jury acquitted the prisoner; but the medical facts of the case rendered it impossible that the mother could have administered the poison—setting aside all absence of motive for such an act. (See *Guy's Hospital Reports*, vol. 4, p. 396.)

In the case of Catharine Wilson, 1862, the property of oil of vitriol to acquire a high temperature in contact with water pointed to the accused as the person who attempted to administer this poison. She gave, while acting as nurse to the wife of a man with whom she was cohabiting, some oil of vitriol in a wine-glass, representing it to be castor-oil. The woman requested that some water might be put to it. This was done, but the glass became suddenly so hot that the woman could not hold it. She put it to her lips, which were burnt by it, but she did not swallow any. The glass was handed back to the accused, who threw the contents away and washed out the glass. But for the clear description of the effect of adding water to the liquid the nature of the poison would not have been known, as after the occurrence no oil of vitriol was found. The prisoner was acquitted on the ground that she might have made a mistake. She was subsequently convicted and executed for poisoning. It is sometimes exceedingly difficult to trace the act of administration. Some years since I was consulted, with the late Mr. Herapath, of Bristol, in the following case: A lady, in a good social position, had had a child by a solicitor. She placed it with a nurse, visited it regularly, and appeared to treat it with great fondness and affection. When the child was some weeks old, it was at various times attacked with sickness, pain, and purging. The child died, and on examining the body for an inquest, Mr. Herapath found a small quantity of arsenic in the viscera, and the usual appearances produced by this poison. There was no doubt the infant had died from the effect of arsenic; but arsenic could not be traced to the food or to the possession of the mother or nurse. There was nothing whatever to lead to the suspicion that the nurse had poisoned the child; the absence of arsenic from the food was clearly proved by a number of collateral facts. The mother had a motive for its destruction, but no arsenic could be traced to her in any way, and the nurse was positive that the mother at no time gave the infant any food or anything to swallow. Farther than this, the nurse deposed that the mother had never been left alone with the infant. It was thought that there had been a chemical mistake, and that arsenic was not the cause of death. The evidence of Mr. Herapath with his report was submitted to me, and my conclusion confirmed his, that the infant had died from the effects of arsenic, administered in small quantity recently before its death.

The case was tried before C. J. Erle, and as the evidence failed to connect the mother with the act of poisoning she was acquitted. Subsequently to the trial, I was informed that the infant was generally worse after each of the visits made by the mother, and that although she gave it nothing, she was observed on each visit to rub its gums with the ends of her fingers. It was suggested, as an explanation of the facts, that she had each time secretly conveyed into the child's mouth a small quantity of arsenic lodged in the hollows of her finger-nails!

Danger of Premature Opinions.—During the examination of a suspected substance, an analyst is often pressed to give an opinion respecting its nature, before the steps of the process are complete. This may arise from the anxiety or curiosity of those who are interested in the proceedings. There is a rule, however, which it appears to me should be always followed on these occasions; that no opinion whatever should be expressed until the *whole* of an analysis is completed. It often happens, in the hands of the ablest analyst, that the last steps of a process lead to a result very different from that which was anticipated at the commencement. The truth is, it is not by one character, but by many, that a poison is identified; and, therefore, a suspicion derived from a few incipient experiments, is very likely to be overthrown by continuing the investigation. In the Boughton case, Dr. Rattray gave an opinion in the first instance, that the poison administered to the deceased was arsenic; but he subsequently attributed death to laurel-water! A case occurred, within my knowledge, in which arsenic was pronounced to be present, when sulphuric acid was really the poison! In a case tried at the Kingston Assizes, a medical witness admitted that, at the coroner's inquest, he stated the poison to be arsenic, but by subsequent experiments he found that it was oxalic acid. In another case, which was the subject of a trial, the poison was at first stated to be oxalic acid, but on a more careful examination it was shown to be arsenic!

A mistake respecting the nature of a poison not merely impedes the course of justice, by throwing a doubt upon evidence which ought to be, beyond all question, clear and satisfactory, but it seriously affects the reputation of a witness. It generally arises from his giving an opinion before he is justified by the facts in so doing. It is, I think, a well-marked line of duty to be pursued on these occasions: 1. That no opinion should be formed from a *few* experiments; and 2. That no opinion should be expressed until the analysis is *completed*. It is obvious that if a man be compelled to admit, in cross-examination at a trial for poisoning, that he has been once mistaken on a question so important, and requiring so decided an answer, a jury may be easily induced to believe that the witness may have made a second mistake, and that his positive opinion is of no more value than that which he first expressed and afterwards retracted. (On the danger of trusting to imperfect chemical analyses, see *Annales d'Hygiène*, 1829, vol. 2, p. 405; vol. 26, p. 399; vol. 29, pp. 103, 474.)

CHAPTER XXI.

EVIDENCE OF POISONING FROM EXPERIMENTS ON ANIMALS—PHYSIOLOGICAL TESTS —FALLACIES—IS THE FLESH OF POISONED ANIMALS POISONOUS?

TOXICOLOGISTS have enumerated experiments upon animals as one among the sources of proof in cases of poisoning. This kind of evidence rests upon the assumption that poisons act in the same manner on man and the lower animals. According to Orfila, this is partially true with only two domestic animals, namely, the dog and the cat—in other cases the results by no means accord. With respect to experiments performed on dogs and cats, I agree with the opinion expressed by M. Devergie (*Médecine Légale*, vol. 2, p. 457), that they are in no case fitted to show the *doses* in which particular poisons are injurious or fatal to man—and they cannot be safely trusted to prove the rapidity of action of different poisons, or the rate of absorption, deposition, and elimination. All that they are fitted for is to enable us to ascertain whether a particular substance be injurious to animal life or not; and, in some cases, its physiological operation on certain organs, as well as the pathological changes produced by it. In Donellan's case, this kind of evidence was received in order to prove the poisonous effects of laurel-water; and in Freeman's case, tried at Leicester in April, 1829, the results of experiments on animals were received as evidence to prove how speedily prussic acid, in certain doses, will destroy human life. The experiments were, however, misleading. The inferences drawn from the results tended to show that the prisoner was guilty of the murder of a woman by administering to her prussic acid; whereas, it was proved by circumstances that he was innocent! An exclusive reliance upon results so obtained, is always liable to lead to erroneous medical opinions. In some experiments made on dogs by Drs. Reid and Simpson they gave *an ounce* of Scheele's prussic acid to one animal; it died in about a minute afterwards. Other dogs of the same size, to which about *six drops* of the same acid, from the same bottle, were given, died in the same period of time; although the dose in the last case was only one-eightieth part of the quantity given in the first experiment.

(*Ed. Med. and Surg. Journ.*, Oct. 1836, p. 500.) From these and similar facts, it is evident that no correct inference respecting a fatal dose of prussic acid in man, or the time required for it to prove fatal, can be drawn from such experiments. Doses so widely differing from each other were found to kill dogs of similar size and strength within the same period of time.

When the question is merely whether a suspected substance administered to another, is or is not poisonous, then we may occasionally be justified in resorting to this kind of evidence, in order to determine the fact—particularly in reference to newly discovered substances which have either not been taken by, or could not be safely administered to human beings. Most of the common poisons of the mineral kingdom are, however, capable of having their presence easily demonstrated by a chemical analysis; and the properties of the substance will be thereby known. But evidence of this description may be sometimes accidentally obtained, and then it may render unnecessary a chemical analysis of the vehicle of the poison; indeed, it may supply proof when no poison is discovered in the body of the deceased. The following is a type of many cases that have occurred in this country. A woman poisoned her husband with arsenic mixed in soup; and after the deceased had made a full meal, she threw the remainder out of a window into a farm-yard, thereby thinking to defeat all attempts at discovering the means which she had adopted to destroy her husband. It happened at the time, that a pig and several fowls were feeding under the window, and they ate up the food as it fell on the ground. The whole of these animals died under symptoms of irritant poisoning. The husband also died. No poison was detected in his stomach, although there were the traces of its action; but on opening the bodies of the animals, the medical witnesses found not only the appearances usually produced by irritant poisons, but arsenic itself was readily discovered in the viscera. This sort of evidence supplied that which was required to complete the case—for while no poison was detected in the body, no portion of the poisoned soup could be procured. The prisoner was convicted and executed.

Good negative as well as affirmative evidence may be sometimes obtained by an examination of the bodies of animals alleged to have been poisoned. In *Reg.* v. *Newton* (Liverpool Autumn Assizes, 1856), it was proved that the prosecutor, to whom, as it was alleged, the prisoner had administered arsenic, went out into a back yard and vomited his food. Some fowls near the spot were observed to be ill during the day, and two died. The prisoner had in the meantime thrown away the remainder of the poisoned food, and washed out the vessels which had contained it. As the prosecutor recovered, there could be no examination of his body, and a portion of the food which prisoner had at the same time prepared for herself, contained no poison. Arsenic was, however, found in the crops of the chickens which had fed at the spot where prosecutor had vomited, and this supplied sufficient proof of the cause of his illness. A woman named Higgins was tried at the Warwick Summer Assizes, in August, 1831, for the murder of her uncle, by poisoning him with arsenic. Her guilt was throughout made very clear. It was proved that she had bought arsenic, and when required to account for the possession of the poison, she said that it was for the purpose of destroying vermin—the excuse resorted to by many criminals. She went, however, farther than this; and actually pointed out, in corroboration of her statement, a dead mouse, which she said had been killed by the poison. This turned out to be an unfortunate part of her defence, for the medical witnesses showed that the mouse had not died from the effects of arsenic.

In the above cases, it will be seen that the evidence from the effects of poison on animals was accidental and ancillary to the main facts of poisoning. There is, however, one instance wherein evidence from experiments on animals cautiously performed, may be of some importance on a criminal trial. I allude to the case in which a poisonous substance is not of a nature readily to admit of a chemical analysis, as for example, certain poisons belonging to the neurotic class. In such a case, if the death of an animal takes place under the ordinary symptoms of poisoning from the administration of a substance, part of which has been taken by the person whose life was thus attempted, the evidence is conclusive. This remark applies to liquids or solids which are made the vehicle of a poison—not to any matters vomited or found after death in the stomach. The results in the latter case would be fallacious; because such matters, without containing any poison, might give rise to vomiting and other symptoms in an animal. The symptoms produced by some poisons, *e. g.*, strychnia, are of such a special character, and the same in all mammalia, that a fair inference may be frequently drawn from the effects produced by this alkaloid. Thus, in the case of *Reg.* v. *Dove* (York Autumn Assizes, 1856), the proof of the presence of strychnia in the stomach of deceased, was partly based on the effects produced on animals by a prepared extract of the contents. A sufficient quantity was procured to kill several animals under the usual tetanic symptoms produced by this poison. The evidence was considered to be conclusive, and more satisfactory than the application of chemical tests to extracts of organic matter containing the poison.

Physiological Tests.—When in a case of alleged alkaloidal poisoning, chemical tests fail to demonstrate the nature of the poison, this kind of evidence may be resorted to in order to corroborate a medical opinion based on symptoms and appearances. A purified extract of a suspected liquid, or of the contents of the stomach, may be introduced into a wound in the neck of a rabbit and the results watched. If symptoms follow corresponding to those of the suspected alkaloids, this would be strongly confirmatory of the fact of poisoning. There is, however, a difficulty which has been suggested, *i. e.*, that these animals are tolerant of certain poisons, *e. g.*, atropia, and therefore a negative result might lead to the erroneous conclusion that no poison was present. On the other hand, does it invariably follow that if an animal dies under these circumstances, this fact furnishes a clear proof that the suspected poison was really present? From the researches of Albertoni and Lussana, it would appear that visceral extracts so procured, are of themselves poisonous without the addition of any poisonous ingredient. (*Ann. d'Hyg.*, 1874, vol. 2, p. 114.)

Taking advantage of the extreme sensibility of the frog to the effects of minute doses of strychnia, the late Dr. Marshall Hall proposed what has been termed the frog-test. A frog is prepared for the purpose, and placed in a bath containing a solution of strychnia, which may even amount to less than $\frac{1}{100000}$th (?) part of a grain. If the poison be present in this small proportion, the animal is said to be suddenly seized with tetanus, and the body and limbs remain rigidly extended. If there be no strychnia the frog is unaffected. Taking this experiment alone, the result would not be sufficient to establish, beyond all doubt, the presence of strychnia, for these animals are readily tetanized by very slight causes, independently of strychnia; and without some corroboration the inference of the presence of this poison from such a result would not, it appears to me, be justifiable. It has been found that morphia produces tetanic symptoms in frogs. The negative evidence which it furnishes would, however, be better than the affirmative; if a healthy frog had not symptoms of tetanus under these circumstances, it would go very far to prove the absence of any trace of that poison which would admit of detection by chemistry. The strychnoscopic test, therefore, must be regarded as an adjunct to other means of research, but not of itself sufficient to produce conviction.

When the food which is supposed to have caused symptoms of poisoning can be procured, this should be employed for the purpose of testing its poisonous or non-poisonous nature in preference to any liquid or solid removed from the body of the deceased. Foderé mentions a case, in which a child, after having partaken of some broth, fell into a state of stupor, lost the power of swallowing, and foamed at the mouth. Some of the meat from which the broth was made was given to a cat. The animal was seized with convulsive fits, alternating with stupor, and died in about five hours. It was rendered probable from the symptoms, as well as from an examination of the body of the animal, that these effects were caused by the introduction of a narcotic plant (hyoscyamus) into the broth. (*Méd. Lég.*, vol. 4, p. 72.) A remarkable instance of this kind of evidence will be found under PHOSPHORUS, in which a shepherd and his dog were poisoned by this substance, which was detected in the stomachs of both after fourteen days' interment. On several occasions similar evidence has been received by an English court of law. A woman named Sherrington was tried at the Liverpool Spring Assizes in 1838, for the attempt to administer poison to one Mary Byres. The evidence showed that the prisoner had sent to the prosecutrix a pudding, by two young children. On the way, these children tasted it, and finding that it had an unpleasant taste, the prosecutrix was put on her guard. The pudding was sent to a surgeon to be analyzed; but he could detect no poison in it. He suspected, however, that it contained a vegetable narcotic poison. He gave a piece about the size of an egg to a dog. In twenty minutes the dog became sick—in forty minutes it lost the use of its limbs—and died in three hours. The prisoner was convicted. Cases in which evidence of this kind, accidentally obtained, has been made available on charges of criminal poisoning, are now very numerous. (See *Reg.* v. *Foster*, Suffolk Lent Assizes, 1847.)

While experiments on animals may give us information on the nature of the symptoms produced by a particular poison, and on the power of discovering it in the absorbed or unabsorbed state in the body, they are not fitted to convey to us any accurate knowledge on the time of occurrence of symptoms, or on the dose required to cause death. In Freeman's case (*ante*, p. 166), the person accused narrowly escaped conviction and execution, by reason of undue confidence placed in experiments on animals, as evidence of the time of accession of insensibility in man, from a fatal dose of this poison.

In the case of Cook (*Reg.* v. *Palmer*, 1856), the danger of relying upon the action of strychnia on animals as evidence of the time for the commencement of symptoms on man, was strongly manifested. In this case no symptoms of strychnia-poisoning had been observed in Cook, until an hour or an hour and a quarter had elapsed after certain pills had been administered to him by the prisoner. Two experts, who appeared for the defence, considered that this was too long a time for the action of strychnia to be delayed, because the poison produced its effects on animals within a much shorter period. One of these (the late Mr. Nunneley, of Leeds) stated that he had experimented with this poison on sixty animals, from dogs to frogs, and the time of occurrence of symptoms varied from two to thirty minutes, the average period for their commencement being about five or six minutes. The other expert stated that the longest interval he had observed in his experiments was three-quarters of an hour. Such experiments only tend to error and confusion. It is now a well-ascertained fact that symptoms of strychnia-poisoning in man have been delayed for two and for three hours; and their appearance is especially delayed when the poison has been taken in the form of hard pills, and the person has fallen asleep after taking them, both of which conditions existed in the case of Cook, but not in the experiments on dogs and frogs!

For the first time, probably, on a trial for murder in this or any country, the counsel for the defence, the late Serjeant Shee, asked permission of the Court that the jury might be permitted to witness the poisoning of a few animals by strychnia, in order that they might draw their own inferences as to the commencement and course of the symptoms, and the period of death. This the Court very properly refused to permit. It is obvious that men in the position of the jury could not be in a condition to draw correct inferences from such experiments. They had already led into a gross error the "expert" witnesses called for the defence, and might have more readily deceived a jury composed of non-professional persons.

Is the Flesh of poisoned Animals poisonous?—This is a question which it is necessary to consider, because poultry and game are not unfrequently poisoned wilfully or accidentally, and in this state they may be eaten unsuspectingly. It is well known that grain is often saturated with a solution of arsenic for agricultural purposes before it is sown: if this grain is eaten by poultry, it will destroy them; and a question may arise as to the effects which the flesh of the animals so poisoned is liable to produce on man. In other instances poison has been placed in the way of these animals, with the malicious object of destroying them. Thus wheat or oats saturated with arsenic, or with that poison intermixed, have been placed in game preserves, for the purpose of destroying pheasants and other birds. In the spring of 1846, two blackcocks were sent to me for examination, from the extensive preserves of a nobleman in Scotland. They had been found dead on the ground. A quantity of arsenic was discovered intermixed with oats and the shoots of the larch, in the crops and gizzards of each bird, and arsenic existed also in the pectoral muscles and soft organs. There had been previously a very large destruction of game on the estate, as it was inferred, from poison. Many similar cases, in which poultry and game were poisoned, have since come before me. Sir R. Christison reports a case which renders it probable that the flesh of poisoned animals is occasionally poisonous. (*On Poisons*, p. 81.) This subject has been examined in reference to sheep by M. A. Guérard. (*Ann. d'Hyg.*, 1843, vol. 1, p. 468.) Some sheep were poisoned with arsenic, and it became important to determine how far their flesh was rendered poisonous and unfit for food. A commission was appointed by the French Academy to make inquiry respecting the facts, and M. Guérard has furnished a summary of the results. The sheep submitted to experiment appeared well, even when they were daily passing arsenic through the fæces and urine. On giving to a young dog the flesh of a sheep which had died from arsenic, the animal after two days was seized with diarrhœa, and arsenic was detected in the fæces and urine. Another dog, which ate the viscera, previously washed, had vomiting and symptoms of a more serious kind; it became thinner, but did not die from the effects of the poison. These results prove that the flesh of poisoned animals is noxious; but if the animals live sufficiently long, the whole of the arsenic is voided in the urine and fæces, and the flesh may then be eaten with impunity. In an experiment on one sheep, arsenic was found in the fæces twenty-two hours after the introduction of the poison into the stomach. Its elimination was daily traced; and fifteen days after it had ceased to appear in the fæces, it was found in the urine. It ceased to appear in the urine on the thirty-fifth day; and when the animal was killed on the thirty-eighth day, no arsenic was found in its body. Six persons ate the flesh without suffering any ill effects, and a dog ate the viscera without manifesting any symptoms of poisoning. The flesh, therefore, is only noxious in the early or acute stage of poisoning, and it is not fit for food until three or four days after arsenic has ceased to appear in the urine. Arsenic and corrosive sublimate are much used in this country as a lotion for the purpose of destroying the fly in sheep. In a case reported by Mr. Annan, two sheep died from the effects of the external application of corrosive sublimate, a poison which is most easily absorbed. (*Med. Times*, July 25, 1846, p. 331.) The flesh of these animals might have proved noxious if it had been eaten.

The flesh of animals poisoned with copper has been known to produce serious effects among those who have eaten it as food. Dr. Galtier relates the following case: A pig, which had been fed with corn soaked in blue vitriol, was so affected that the owner had it killed, and sold the carcase to a butcher. Seventeen persons who ate of the flesh of this animal were seized with violent colic, and those who ate the blood made into black puddings, also suffered severely. The milk of a goat, which had eaten sour food out of a copper vessel, occasioned nausea, vomiting, colic, purging, cramp, and other alarming symptoms, among fifteen persons who partook of it. They had before taken milk from the same goat without injurious consequences. The animal itself became ill, and died on the third day, under all the symptoms of poisoning. The mucous membrane of the small intestines was found inflamed. (*Toxicologie*, vol. 1, p. 631.) It is to be regretted that no analysis of the food was made in these cases of acute poisoning (p. 55, *ante*).

But little is known concerning the effects produced by absorbed organic poisons on the flesh of animals. It might be supposed that a few experiments with such a poison as strychnia would at once supply an answer to the inquiry. I have frequently seen the larvæ of insects grow and thrive on the flesh of animals killed by strychnia; but then, on experiment, I found that the larvæ were not killed by the application of a strong solution of sulphate of strychnia to their bodies, and they did not appear to be in any way affected by a dose of the poison which would have speedily destroyed a rabbit. Dr. Harley found that the flesh of animals killed by minimum doses of strychnia did not act as a poison to other animals. He fed a hedgehog on poisoned flesh for a period of fourteen days, without being able to detect the slightest symptom of poisoning. The poison must, therefore, he concludes, have been either decomposed, or it was not present in sufficient quantity. (*Physiological Action of Strychnia*, p. 15.)

Dr. Macadam states that he killed a horse with thirty-two grains of strychnia, given at intervals. He fed a large-sized terrier dog for two weeks on the flesh of this horse; the animal eating every day during this period two pounds of muscle. The terrier dog, he says, lived and throve on the flesh, and did not betray the faintest shadow of tetanic symptoms. He states that, on analysis, he found distinct evidence of strychnia in the muscle and blood of the horse, but the quantity is not stated. (*Pharm. Jour.*, August, 1856, p. 124.)

Bernard gave as food to dogs the flesh of rabbits which had been poisoned with strychnia and nicotina, and the dogs experienced no ill effects. When, however, a larger dose of poison than was sufficient to cause death had been given, then the surplus acted fatally on other animals. This would be, in fact, a case of direct poisoning. But the flesh alone did not appear to have had any poisonous action, the quantity of absorbed poison contained in it having been too small. The intestines of animals poisoned with strychnia and nicotina have proved fatal to other animals. (*Leçons sur les Effets des Substances Toxiques*, 1857, p. 281.) From these observations, therefore, it appears that it is not the absorbed, but the unabsorbed, portion of this alkaloidal poison, which is likely to affect other animals.

The results obtained by Drs. Harley and Macadam, as well as by M. Bernard, bear out the view that the flesh of game killed by the arrows of certain Indian tribes in South America, poisoned with woorara (*curara*) may be eaten with impunity. The quantity actually absorbed and diffused through the flesh is, as in the case of strychnia, either too small to cause symptoms of poisoning, or the organic poison itself undergoes some change in the body, by which its noxious effects are destroyed.

Dr. Livingstone states that the flesh of animals killed by the arrow-poison of the Central Africans is eaten by the natives with impunity. The poison (*kombi*) soon takes effect on the animal. It produces muscular paralysis and finally paralysis of the heart. The animal drops. A portion of flesh around the wound is cut away and all the rest is eaten. (Fraser, *On the Kombi Poison.*)

Dr. Fayrer (*Thanatophidia of India*) observes that the flesh of an animal dead from snake-poison is not affected by it; animals and men eat the flesh with impunity. His servants ate all the fowls killed in his experiments by the bite of the cobra. It would appear that but little, if any, of this poison is deposited in the tissues. It is chiefly contained in the blood. In the case of poisoned animals, the amount of absorbed poison appears to be too small to render a small quantity of flesh poisonous as food.

The following cases, of which a note was sent to me by a friend in Australia, shows, however, that the flesh of animals may be, on some occasions, the means of transferring poison to human beings. In April, 1871, a family, consisting of nine persons, varying in age from seventy years to an infant, were attacked with symptoms resembling those of irritant poisoning after eating some mutton which had been purchased of a butcher in the usual way. The symptoms were violent vomiting and purging, pains in the limbs, and great prostration of strength. It was found that other families had suffered from eating mutton under similar circumstances. An inquiry was instituted by the authorities, and it was found that the sheep, in travelling to Melbourne, had been fed on the wild cucumber (colocynth), the native melon, and the lotus Australis, all of them poisonous plants, which abounded in the colony. Some sheep died within half an hour of their feeding on these plants. Others were killed and the meat was sold without exciting any suspicion that it was poisoned.

It is a curious fact, that the bodies of animals may, in some instances, be made the vehicles of transferring poison to the human subject, while the animals themselves do not appear to suffer from its effects. Thus the flesh of the pheasant, which feeds on the buds of the *Kalmia latifolia*, in North America, is deemed poisonous during the winter and spring. (*Beck's Med. Jur.*, p. 854.) The flesh of hares which have fed upon *Rhododendron chrysanthemum*, is considered to be poisonous. A singular case occurred in France, in which a whole family near Toulouse was poisoned with a dish of snails. The symptoms under which they suffered were those of narcotico-irritant poisoning; and it was found, upon inquiry, that the snails had been gathered from bushes where they had fed upon the leaves and young shoots of the *Coriaria myrtifolia*, a vegetable poison. (*Gaz. Médicale*, Oct. 1842; also *Med. Gaz.*, vol. 31, p. 237.)

Dr. Dumas, of Cette, has described a similar set of cases which occurred under his observation in April, 1873. Seven persons in a family had partaken of a dish of snails. All were seized with symptoms of irritant poisoning about fifteen or twenty hours after the meal, and in a degree proportioned to the quantity eaten by each. There were nervous symptoms indicated by headache, giddiness, and delirium. On inquiry it was found that the snails had been collected from shrubs such as the box, or a species of euphorbia and other plants of a noxious kind. It was observed that the snails had a very bitter taste. One theory set up was that they had absorbed and transferred the poison from the vegetables, and another that, like marine mollusca, they might themselves have acquired poisonous properties. (*Ann. d'Hyg.*, 1874, vol. 1, p. 446.)

It has been long known that honey derived from bees which have fed upon the *rhododendron*, *kalmia*, *azalea*, or *datura*, growing in certain districts, acts as a narcotico-irritant poison, producing giddiness, vomiting, and purging. It is stated that arsenic has found its way into honey under the following circumstances: On the line from Cologne to Elberfeld there is an arsenical factory, and it has been observed that the honey collected round this spot has caused symptoms of poisoning among those who have eaten it. On analysis, the honey was found to be impregnated with arsenic. (Bouchardat, *Ann. de Therap.*, 1874, p. 207.) As arsenic is a poison to insects, it is difficult to understand how the bees which produced the honey should have escaped the effects. In the chapter on ANIMAL IRRITANTS some other facts will be mentioned, from which it would appear that the *milk* of cows fed in certain districts of America is poisonous, and gives rise to serious symptoms, whether taken as milk or made into cheese. The flesh of the animal possesses also poisonous properties; while the animal itself does not suffer in health from feeding on the plants. These facts are explicable on the supposition that there are specific idiosyncrasies among different classes of beings, thus rendering what is innoxious to one a poison to another.

CHAPTER XXII.

MORAL AND CIRCUMSTANTIAL EVIDENCE IN POISONING—STATISTICS OF POISONING IN ENGLAND AND WALES—CASES ADMITTED INTO GUY'S HOSPITAL—COMPARATIVE DEATHS FROM POISON.

THE duty of a medical witness, as such, is accomplished when he has proved, on a charge of criminal poisoning, that death was *certainly* due to poison. The moral and circumstantial evidence must show that the accused was the party who gave it;—this proof often fails—the fact of administration cannot be brought home to the accused, and the case falls to the ground. It is not within the province of this work to treat of moral and circumstantial evidence in cases of poisoning. Proofs of this kind, it is true, are sometimes very closely mixed up with the evidence of professional witnesses, and in the foregoing chapters some of these have been already adverted to. A witness must, however, be cautious not to base his opinion, in questions of poisoning, on moral and circumstantial proofs. He is called upon to give a medical opinion of the cause of death, from *medical* facts only. The moral and circumstantial facts refer chiefly to the recent possession or purchase of poison without reasonable cause—motives for acts of poisoning—the opportunity of administering—and the actual administration of poison by a particular person—with false reasons assigned for conduct. These circumstances unexplained, with other facts, showing criminal design or intention, are the material points on which a charge of murder rests, irrespective of medical evidence. It is within the province of a jury alone to decide on the relevancy and value of these portions of the evidence against an accused person, although it must be confessed that many of the facts can be properly estimated only by persons versed in medical science.

Supposing death by poison to have been clearly proved, it may be necessary to ascertain whether the act was the result of *accident, suicide,* or *homicide.* This is a question also for a jury to determine, and not for a witness; although its solution often depends upon a proper appreciation of medical circumstances. Suicide or murder may sometimes be inferred, according to the medical evidence given of the effects of certain poisons. Some speedily annihilate volition and the power of locomotion, and therefore render it a question of serious difficulty whether particular acts could or could not have been performed after the deceased had taken the poison. On the answer to this question may depend the acquittal or conviction of a person charged with the crime.

There is one peculiarity in the legal consequences of the act of killing by poison, namely, that the act itself is generally considered in law to furnish distinct evidence of malice. If a poison is knowingly administered to another, with the intention of destroying life, the crime is never reduced to manslaughter—whatever may have been the provocation which the party administering has received from the person whose life he has thus taken. It is not necessary, therefore, that any particular enmity should be proved to have existed between the prisoner and deceased, although this when proved may weigh as a strong moral circumstance against the former. The absence of any apparent motive for the crime, on the other hand, is always regarded as a strong presumption in favor of the accused. When a man dies from a wound received in a quarrel, the law will sometimes find an excuse for the act, from the heat and passionate excitement under which the aggressor was laboring at the time; but if the aggressor should avenge himself by secretly administering poison to his adversary, there is no excuse for the act, since there has been time for reflection, and the act evinces cool and deep-rooted malice. That death by poison should ever amount to manslaughter, therefore, it must be shown that the substance was administered to, or laid in the way of the deceased by mistake, or with innocent intention; and the proof of this always lies with the accused—the law inferring that malice exists until the contrary appears from the evidence. Whether malice exists or not is, however, in general soon made apparent from the evidence for the prosecution. There are many cases in which the act of killing by poison arises from gross negligence, as where a man, dispensing medicines, weighs out arsenic for calomel, or strychnia for bismuth, or dispenses laudanum for black draught. Such cases, which have been of frequent occurrence, have been hitherto treated with too great leniency. The persons inculpated have generally been discharged on the ground of misadventure. "Killing by negligence" has been hitherto treated as manslaughter; but it is now proposed by statute to make it a distinct crime, and to assign to it a special punishment.

In relation to medico-legal practice, the *statistics* of poisoning are of some interest. They indicate to a medical jurist the substances which are most frequently selected for the purposes of suicide and murder, and with the properties of which it will be expected that he should be acquainted. Unfortunately, very few tables of this kind have been published; and those which have appeared are defective in many points. One of the most complete is that which was published by order of the House of Commons from returns made by the coroners of England, of the number of inquisitions held during the years 1837 and 1838, wherein death was caused by poison. The following is an abstract of this return, which appeared in the *Medical Gazette* for November, 1839.

The number of deaths by poison (returned) in the two years abovementioned, were, exclusive of two cases of suffocation by gases, 541. The substances which caused death may be taken in the following numerical order:

Opium	Laudanum,	133		
	Opium,	42		
	Other preparations,	21	196	
Arsenic,			185	
Sulphuric acid,			32	
Prussic acid,			27	
Oxalic acid,			19	
Corrosive sublimate and mercury,			15	
Mixed or compound poisoning,			14	
Oil of bitter almonds,			4	
Poisonous mushrooms,			4	
Colchicum, nux vomica (of each 3),			6	
Nitric acid, caustic alkali, tartar emetic, acet. morphia, strychnia, deadly nightshade, aconite (of each 2),			14	
Bichrom. potash, nit. silver, Goulard's extract, sulph. iron, mur. tin, hellebore, castor-oil seeds, savin, hemlock, cantharides, cayenne pepper (of each 1),			11	
				527
Unknown,				14
				541

[A little discrepancy exists in the relative number of cases, probably owing to the fact that in several instances some of the poisons were taken in a compound state. The reader will find the details in the *Med. Gaz.*, vol. 25, p. 204.]

It will be seen from this table that the largest proportion of cases of poisoning in England and Wales are those with *opium* and *arsenic;* the greater number of the former being cases of suicide and accident, and of the latter cases of criminal poisoning. There can be no doubt that the deaths from poison which annually occur in England and Wales are much greater than this table represents. The annual registration of deaths, although defective with respect to the number of individual poisons—two only, *i. e.*, arsenic and opium being commonly recorded, and these imperfectly—shows that the mortality from this cause, including overdoses of medicine, is greater than is commonly supposed. I here subjoin a table of the deaths from poison in 1840, drawn up from the Sixth Annual Report of the Registrar-General (1844). The deaths from this cause in 1840 are stated to have been 349, of which number there were 181 males and 168 females. The cases of suicide from poison were 161, being 87 females to 74 males; the cases of accident or homicide were 188, being 107 males to 81 females. Of the 75 cases of poisoning by opium, 42 occurred in children under five years of age—a lamentable proof of the extensive mortality among children from the improper administration of this drug. These cases occur among the returned deaths from opium; but under the head of medicines improperly administered, three-fourths of the deaths took place among children under five years of age!

Opium,	75
Arsenic,	32
Other poisons, including medicines improperly administered,	242
Total deaths from poison in 1840,	349

It would be a considerable benefit to medical science if the poisons which caused death were correctly specified in the Registration returns. The poison or medicine should be stated as distinctly as in the returns now made with respect to fatal diseases. The cases of poisoning with arsenic and opium in the above table, are evidently understated.

A proper collection of hospital statistics would throw great light on the frequency of death from poison, and the nature of the poison taken. Mr. Wilson (*The Bane and the Antidote*, Birmingham, 1856), states that, in sixty-three cases of poisoning treated in the Birmingham Hospital from 1848 to 1856, a period of eight years, the nature of the poison used was as follows:

Opium or laudanum, 12; sulphuric acid, 9; oxalic acid, 8; acetate of lead, 5; nitric acid, 4; arsenic, 3; sulphate copper, 2; iodine, 2; nux vomica, 2; ammonia, 2; cyanide potassium, 1; corrosive sublimate, 1; strychnia, 1; essence of bitter almonds, 1; muriate of morphia, 1; charcoal-vapor, 1; other poisons, 8; total 63. The deaths were only 5.

This table indicates that the number of deaths from poison are few compared with the actual cases of poisoning. Probably, on an average, not more than one in five cases proves fatal.

I am indebted to Dr. Steele for some statistical information on this subject, as collected from the records of Guy's Hospital. During the years 1854-5-6, there were admitted into the hospital 27 cases of poisoning, out of which number 11 proved fatal. The poisons used were as follows:

Arsenic, 2; sulphuric acid, 3; nitric acid, 1; oxalic acid, 4; laudanum, 5; prussic acid, 1; oil of bitter almonds, 1; solution of ammonia, 1; brandy, 1; chloride of zinc, 1; poisonous berries, 2; locust beans, 1; oxide of mercury, 2; unknown, 2; total, 27.

Of the sixty-three cases reported from the Birmingham Hospital only five proved fatal. This is in the proportion of about 8 per cent., or one death for twelve cases. Among the twenty-seven cases at Guy's Hospital, there were eleven fatal cases, making a proportion of 40 per cent., or two deaths for every five cases. The average annual admissions at the Birmingham Hospital were eight; at Guy's Hospital nine cases.

Dr. Steele has furnished me with the subjoined report of cases admitted into Guy's Hospital during fourteen years, from 1860 to 1874. The total number of cases was 119, of which 21 proved fatal. The poisons used were as follows:

Arsenic, 9; chloride of zinc, 4; sulphuric acid, 5; hydrochloric acid, 10; white and red precipitates of mercury, 11; oxalic acid, 14; nitric acid, 2; prussic acid, 1; sugar of lead, 13; carbolic acid, 2; phosphorus, 1; bichromate of potash, 2; turpentine, 1; oil of hartshorn, 5; opium, 18; nux vomica, 2; belladonna, 3; atropia, 1; paraffin (petroleum), 3; mushrooms, 1; syrup of poppies, 1; sulphate of indigo, 1; croton seeds, 1; camphor, 1; mussels, 1; foul air, 4. To these may be added 31 cases of alcoholism (poisoning with alcohol); they all recovered under treatment.

According to a paper in the *Ann. d'Hygiène* (1873, vol. 1, p. 240), there were in Finland, from 1860 to 1866, thirty fatal cases of poisoning, namely, from arsenic, 7; phosphorus, 5; nux vomica and strychnia, 4; corrosive sublimate, 2; sulphuric acid, nitric acid, potash, chromate of potash, prussic acid, petroleum, opium, morphia, lycopodium, of each one case, making a total of 9, and in 3 cases the poison was not determined.

Mr. Wilson published an analysis of the *deaths* from poison in England alone, taken from the Registrar-General's Report for six years, from 1848 to 1853—the latest returns which had then been published. The total deaths, as recorded in these returns during the period abovementioned, were 3218, distributed as follows:

	Males.	Females.	Total.
1848,	308	261	569
1849,	290	236	526
1850,	304	249	553
1851,	275	253	528
1852,	253	300	553
1853,	270	219	489
Total deaths in six years,	1700	1518	3218

This gave at that date an average annual mortality of 536 deaths from poison in England alone. From the table of returns, elsewhere published (*ante*, p. 176), it will be perceived that, while the deaths from poison in two years (1837–8) for England and Wales, amounted to 541, or an annual average of 270, in 1848, *i. e.*, ten years later, the number of deaths amounted to 569. In the five years from 1863 to 1867, the annual average was 418, and in the following year, 1868, the number recorded was 406. So far as these returns will allow a judgment to be formed, the deaths from poison actually decreased in the twenty years ending with 1868; and the apparent increase between 1838 and 1848 may be in a great measure referable to the increase of population.

But cases of poisoning cannot be estimated only by those which prove fatal to life. There are many who suffer temporarily and recover from the effects. Assuming the average mortality to be not more than one out of three cases, this would give a total of 1600 cases of poisoning per annum. The relative proportions in which the deaths are occasioned by the various poisons may be computed from the coroner's returns. In those for 1837–8, the poison was ascertained in 527 cases. Of these opium and its preparations formed 37 per cent.; arsenic, 35 per cent.; sulphuric acid, 6 per cent.; prussic acid, 5 per cent.; oxalic acid, 3.5 per cent.; and mercurial preparations about 3 per cent. Opium and arsenic were the causes of death in three-fourths of the whole number of cases, and opium caused a greater number of deaths than arsenic. It appears that in France about two-thirds of the cases of poisoning over a period of twenty years, were caused by arsenic. (Flandin, *Des Poisons*, vol. 1, p. 448.)

The following table, issued by the Registrar-General in June, 1869, shows the number of deaths recorded at a more recent period. In five years, 1863–7, it is stated that there were 2097 deaths from poison in England and Wales. The poisons were not described in 477 cases, and among some of those recorded there were apparently errors, especially respecting the deaths alleged to have been caused by the salts of lead, of which the number was set down at 242! In 50 cases the deaths were assigned to improper medicine and unwholesome food. These deductions leave 1270 cases in which the substance causing death was ascertained:

Opium—laudanum, syrup of poppies, and Godfrey's cordial,	482
Opium and its compounds,	114
Morphia,	82
Prussic acid, cyanide potassium,	151
Essential oil of almonds,	31
Arsenic,	88
Oxalic acid,	66
Strychnia and vermin killer,	61
Mercury (compounds of),	58
Sulphuric acid,	53
Nitric acid,	16
Hydrochloric acid,	8
Carbolic acid,	5
Alcohol.	35
Phosphorus,	15
Ammonia,	11
Chloride zinc,	8
Mussels,	8
Fungi,	6
Aconite,	6
Belladonna,	6
Chlorodyne,	4
Turpentine,	3
Colchicum,	3
Nitre,	3
Sulphate of copper,	3
Cantharides,	2

This table would lead to the conclusion that the deaths from poison eight years since, amounted to an annual average of 528; but in three-fifths of these cases only, was the nature of the poison distinctly ascertained. There are no doubt many cases of death from poison which are registered under the head of fatal diseases, as cholera, convulsions, gastric fever, disease of the heart, apoplexy, epilepsy, etc. If proof of this were required, I might point to the numerous cases of exhumation in which it has been shown that death has taken place by poison, while the bodies had been buried, under certificates of death from disease, and registered accordingly. These are less numerous than they formerly were, but the recent occurrence of such a case as that of Mary Ann Cotton (*Reg.* v. *Cotton*, Durham Lent Assizes, 1873), necessarily gives rise to an uneasy feeling in the public mind regarding secret murder. This woman was convicted of the murder of her stepson by poison a year before. The deceased had been buried under a certificate stating the cause of death to be gastric fever. When the exhumed body was examined arsenic was detected in it, and this proved to be the sole cause of death. This woman, it was stated upon well-ascertained facts, had at different times killed her mother, fifteen children, three of her husbands, and a lodger, making altogether *twenty persons* in a few years! They died rather rapidly one after another, and the medical certificates represented the cause of death as gastric fever, when in some of the cases the symptoms were not at all consistent with this disease. The motive for these murders was to obtain the small premiums for which the lives of the deceased had been secretly assured by the prisoner herself! Such cases could not occur, if there were more perfect methods of observing and recording the causes of death.

IRRITANT POISONS.

MINERAL IRRITANTS.

ACID POISONS.

CHAPTER XXIII.

Oil of vitriol or sulphuric acid—Symptoms—Effects of the concentrated and diluted acid—Death from asphyxia—Time at which the symptoms begin—Power of locomotion—Remission of symptoms—Appearances in the dead body—Acute and chronic cases—Fatal dose—Period at which death takes place—Treatment.

Irritant poisons comprise a large number of substances derived from the mineral, vegetable, and animal kingdoms. They include a number of acids, alkalies, non-metallic, and metallic substances, differing widely from each other in physical and chemical properties.

They are identified by the physiological characters elsewhere assigned to the class (*ante*, p. 74). They irritate and inflame, and sometimes corrode and destroy the parts of the body with which they are brought in contact. The mineral poisons may be, for convenience, arranged in four groups, comprising acids, alkalies, non-metallic, and metallic substances. Among the acids will be included a few derived from the organic kingdom.

ACIDS here imply those solid or liquid substances which have a sour taste, redden blue litmus-paper, and are neutralized by alkalies and form salts. One of the most delicate tests for acidity or the presence of an acid in a liquid is a solution of the ammonio-chloride of silver, which gives a white precipitate. The *mineral* acids will first require consideration.

OIL OF VITRIOL OR SULPHURIC ACID.

This is met with in commerce in two states, either concentrated or diluted. The concentrated acid is a heavy oily-looking liquid, often of a brown color; it has a strong, sharp, acid taste—it powerfully reddens vegetable colors, and corrodes and destroys most kinds of organic matter. The term, oil of vitriol, is strictly applied only to sulphuric acid which has an oily consistency and a great specific gravity (from 1.800 to 1.845). It was so called because it was obtained by the distillation of green vitriol, of which it was considered to be the oil or spirit. It is in this state eminently corrosive, and this corrosive property is lost when the oily consistency is removed by dilution with its bulk of water. Oil of vitriol is, under all circumstances, sulphuric acid, but sulphuric acid is not in all cases oil of vitriol. The question has been raised whether an acid of a sp. gr. of 1.420 should be regarded as oil of vitriol. Such an acid contains 56 per cent. of water; it has no oily consistency, and has none of the well-marked chemical (corrosive) properties of oil of vitriol.

Sulphuric acid is frequently taken as a poison by suicides; but probably there is no case in which the sufferings of a person before death are more intense. In medico-legal practice it is not common to find that this acid is employed for the purpose of murder. Children have, however, been destroyed by a quantity of it being poured down the throat; and it is obvious that a person who is drunk or asleep may be thus easily killed. With these exceptions, which are of rare occurrence, instances of fatal poisoning by sulphuric acid may be pretty equally divided into cases of suicide and accident. The taking of this liquid is a frequent form of self-destruction among females; less frequent among males, and by no means uncommon as an accident among young children of both sexes. On the discovery of a dead body, poisoned by sulphuric acid, a medical jurist will have, then, especially to consider the age of the deceased. If it be a new-born child, or an infant, it is certain that the poison has been homicidally or accidentally administered; if a child, all other circumstances being equal, that it has been swallowed by accident; if an adult, that it has been voluntarily taken for the purpose of suicide. It is to be observed that there is no poison which can be obtained more readily or without exciting less suspicion than sulphuric acid, since it is used for many domestic purposes. The only probable case of murder by this poison in an adult, would be where the person was either intoxicated or asleep when it was administered; but even then the individual would be immediately roused. It is not easy to imagine that a criminal, who wished to destroy the life of another, would attempt this by causing him to swallow forcibly a quantity of oil of vitriol, when there are so many other more ready, secret, and speedy means of destruction at hand. It is also impossible that such a substance as this should, like arsenic, be secretly administered in articles of food. Its powerfully acid taste in the smallest quantity, and the fact that the physical qualities of the food would in general be changed by the chemical action of the acid, would certainly lead to a discovery and frustrate the attempt. There are but few instances in which such an attempt to poison has been made. In one of these, a boy being offended with his mistress, put a quantity of common diluted vitriol into a cup of tea which she was about to drink; in another, the attempt to administer was made by putting the acid into coffee. In both instances the taste immediately led to the discovery of the attempt.

Symptoms—The Concentrated Acid.—When this poison, which is one of the most powerful corrosives, is swallowed in a concentrated form, the symptoms produced come on *immediately*, or during the act of swallowing. There is violent burning pain extending through the throat and gullet to the stomach—the pain is often so severe that the body is bent. There is an escape of gaseous and frothy matter, followed by retching and vomiting, the latter accompanied by the discharge of shreds of tough mucus, and of a liquid of a dark coffee-grounds color, mixed or streaked with blood. The mouth is excoriated, the lining membrane and surface of the tongue white or resembling soaked parchment—in one instance the appearance of the mouth was as if it had been smeared with white paint; after a time, the membrane acquires a gray or brownish color; the cavity is filled with a thick viscid phlegm, rendering speaking and swallowing very difficult. If the poison has been administered by a spoon, or the vial containing it has been passed to the back of the throat, the mouth may escape the chemical action of the acid. A medical witness must bear this circumstance in mind, when he is called to examine an infant suspected to have been poisoned by sulphuric acid. Around the lips, and on the neck, may be found spots of a brown color from the action of the acid on the skin. There is great difficulty of breathing, owing to the swelling and excoriation of the tongue and throat; and the least motion of the abdominal muscles is attended with increase of pain. The abdomen is distended and tender. These symptoms, although peculiar and well marked, have been sometimes mistaken for those of disease. (Henke, *Zeitschrift der S. A.*, 1843, vol. 2, p. 284.) The stomach is so irritable that whatever is swallowed is immediately ejected, and the vomiting is often violent and incessant. In a case which occurred to the late Dr. Geoghegan, the patient (a woman) vomited for three or four hours. This symptom then ceased, and did not reappear, although she lived thirty-one hours. (*Med. Gaz.*, vol. 48, p. 328.) Vomiting, although a common symptom, is not always immediate. A case is reported in which a man, æt. 30, swallowed two ounces of oil of vitriol (1.842), and died in twenty-five hours, retaining his senses until the last. Half an hour after he had taken the poison he resembled a patient in the collapsed stage of cholera. The inside of the lips, tongue, and throat were swollen, and had the appearance of being smeared with thin arrowroot. He suffered severe pain, but did not vomit until three-quarters of an hour had elapsed; the vomiting appeared to be then excited by the liquid given to him. The vomited matters were dark, bloody, and viscid. This case is remarkable in the fact that vomiting was not immediate; that there were no spots on the outside of the face; that the poison was swallowed in large quantity on an empty stomach; and there was free voluntary exertion, as, twenty hours after he had taken the poison, the man got out of bed and sat on a night-stool. (*Ed. Monthly Jour.*, 1850, p. 538.)

The matters *first* vomited generally contain the poison; they are acid, and if they fall on a limestone pavement there is effervescence, if on colored articles of dress, the color is sometimes altered to a red, or (if logwood) yellow—the color is discharged and the texture of the stuff destroyed—on a black cloth dress, the spots produced by the concentrated acid are brown, with a fringe of red, and they remain moist for a considerable time. An attention to these circumstances may often lead to a suspicion of the real cause of the symptoms, when the facts are concealed. In a case of attempted murder by sulphuric acid in beer, the nature of the poison was suspected from the beer having corroded an apron on which a portion had been accidentally spilled. After a time, there is great exhaustion, accompanied by general weakness; the pulse is quick and small; the skin cold, and covered with a clammy sweat. There is generally great thirst, with obstinate constipation of the bowels; should any evacuations take place, they are commonly either of a brown or leaden color,—in some instances almost black (carbonaceous), arising from an admixture of altered blood. They also contain corroded portions of the lining membrane of the bowels. There are sometimes convulsive movements of the muscles, especially of those of the face and lips. The countenance, if not livid from obstructed respiration, is pale, expressive of great anxiety and of dreadful suffering. The intellectual faculties are quite clear, and death usually takes place very suddenly, in from eighteen to twenty-four hours after the poison has been taken.

When the acid is *diluted*, the symptoms are of the same character, but less severe, and not so quickly produced. They vary according to the degree of dilution, the poison acting only as an irritant when much diluted. The vomited matters are not so dark-colored; in one instance they were nearly colorless. It may be proper here to state that the diluted sulphuric acid of the British Pharmacopœia has a sp. gr. of 1.094. Six fluid drachms correspond to 40 grains of anhydrous sulphuric acid. The corrosive properties of the acid are destroyed by dilution, but its irritant properties are retained.

Within what period of Time do the Symptoms commence?—Most toxicologists, including Orfila (*Toxicologie*, vol. 1, p. 83, 1843), Christison

(*On Poisons*, 4th ed., 90), and Galtier (*Traité de Toxicologie*, vol. 1, p. 121, 1845), state that the symptoms commence *immediately*, or during the very act of swallowing, *i. e.*, a sense of heat is experienced, with excoriation and burning pain in the throat and stomach. Considering the powerful chemical action of the poison on the thin mucous membrane of the mouth and fauces, it is not easy to understand how there should be any delay in the production of some visible symptoms. In rabbits I have always observed instantaneous effects on the contact of the acid, such as foaming and frothing at the mouth, with a milky-white appearance from the action of the poison on the lining membrane. In most cases that have hitherto been accurately noticed from the commencement, *i. e.*, from the act of swallowing, there has been at first an escape of gas, with severe retching, followed within a short period by vomiting. The question relating to the time of occurrence of symptoms was of some importance at the trial of the *Queen* v. *North* (Guildford Summer Ass., 1846); for upon the answer rested, in some measure, a charge of murder. (*Guy's Hospital Reports*, October, 1846, p. 396.)

From a case observed by Orfila, it appears that even when moderately diluted, there is no delay in the appearance of the symptoms produced by this acid. A man swallowed a certain quantity of sulphuric acid, diluted with its weight (*i. e.*, with twice its bulk), of water, and experienced immediately the most severe suffering. (*Toxicologie*, vol. 1, p. 96.) The common opinion of toxicologists, that this poison, from its local chemical action, produces at once certain effects, is, I believe, correct in all cases in which it is not much diluted with water. It causes some immediate symptoms, and in most cases early vomiting; a fact borne out by the results of repeated experiments on animals. A case is reported in the *Medical Gazette* (vol. 39, p. 147), from which it appears that half a teaspoonful of sulphuric acid was given by mistake for castor oil to a child a year old, and that certain symptoms immediately followed: the child cried and was restless, and this led to the discovery of the mistake.

The local action of sulphuric acid on the mouth, throat, and gullet is generally very energetic; the lining membrane is stripped off in shreds, or peels off in large masses. In a case mentioned by Sobernheim, the lining membrane of the mouth, tongue, and throat came off in one mass. In another, related by Dr. Wilson, the patient, during a violent fit of coughing, brought up a large piece of sloughy membrane, which was found to consist of the inner coat of the gullet much thickened and very firm in texture. Its length was eight or nine inches, and its width that of the gullet; it was of a cylindrical form, and pervious throughout its whole extent. (*Med. Gaz.*, vol. 14, p. 489; also vol. 22, p. 76.) This has been observed to occur in other cases. (See Galtier, *Toxicologie*, vol. 1, p. 199.)

The acid may not go farther than the entrance of the throat. In May, 1857, a case of this kind was admitted into Guy's Hospital. In fourteen days the man left the hospital with his mouth and throat restored to their natural condition. (*Guy's Hosp. Rep.*, 1859, p. 188.)

This poison may destroy life without reaching the stomach—a fact observed in children. The larynx is then acted on—the air-passage is closed by the swelling of the surrounding parts, and the child dies suffocated. In such cases death takes place very rapidly. I have found that rabbits, to which this poison was given, died from this cause in the course of a few minutes. Owing to this local action on the air-passages, sulphuric acid may easily cause death by suffocation. (See case by A. T. Thomson, *Lancet*, June 10, 1837.) E. B., a child æt. seventeen months, was brought into Guy's Hospital in July, 1857. Shortly before, the mother had given it a teaspoonful of oil of vitriol by mistake, for syrup. As it was in a state of apparent suffocation, tracheotomy was performed by Mr. Forster. The child was apparently relieved; but it died in seven hours after taking the poison. (*G. H. Rep.*, 1859, p. 137.)

Death may take place, on these occasions, from an affection of the larynx alone; the acid may not even have reached the gullet (See *Ed. M. and S. Jour.*, vol. 49, p. 583; also *Med. Chir. Rev.*, vol. 28, 399.) A remarkable instance of this kind has been reported by Sir W. Gull (*Med. Gaz.*, 1850, vol. 45, p. 1102). A lady was found dead in a chair holding in her right hand a small vial labelled sulphuric acid—poison. The body was in an easy attitude, half recumbent, and there had been no struggling or motion after the acid had been taken. The air-passages, including epiglottis, larynx, and trachea, showed marks of corrosion; the acid had passed into both lungs, had corroded them, and by acting on the subjacent ribs had formed a crust of sulphate of lime upon the lungs. The large bloodvessels were full of solid charred blood resembling blacking. The lining membrane of the gullet and stomach was uninjured. None of the acid had passed into the stomach. Death had taken place from suffocation, and as the body was not inspected for six days, the acid may have produced much chemical action after death. Owing to this local action on the air-passages, cyanosis (blueness of the skin) has been occasionally observed among the symptoms. (Galtier, *Toxicologie*, vol. 1, p. 192.) Thus, then, as a medico-legal fact of some importance, it is certain that this poison may destroy life without reaching the stomach. There are at least two instances on record in which the acid has destroyed life in consequence of its having been injected into the *rectum* by mistake for a clyster. In one of them, the patient suffered the most acute pain, and died in the course of a few hours. (*Med. Gaz.*, vol. 17, p. 623; *Annales d'Hyg.*, 1846, vol. 1, p. 366.)

The severe pain produced by a large dose of this poison is in many cases sufficient to deprive a person of the power of motion. The patient rolls on the ground in agony. Nevertheless, numerous well-observed facts prove that he may sometimes retain astonishing self-command. In the case of Mr. Schwabe, who died in twenty-four hours from a dose of six drachms of sulphuric acid, it was proved that the deceased, after having swallowed the acid, beckoned to a cabman, got into a cab, and told him to drive to his house as fast as he could. The deceased had at the time a handkerchief to his mouth, and the only circumstance noticed by the driver was, that he looked very pale. (*Med. Gaz.*, vol. 36, p. 826.) A case is quoted by Dr. Galtier, in which a man, æt. 52, after having taken some soup, swallowed three ounces of commercial sulphuric acid. He threw himself upon his bed, and it was not until between three and four hours afterwards that the severe pain which he suffered compelled him to seek for assistance. He got up, dressed himself, and was conveyed to the hospital, where he died five hours after swallowing the poison. (*Toxicologie*, vol. 1, p. 189.) In another case, a child, æt. 9, swallowed an ounce of oil of vitriol, and although instantly seized with severe pain in the throat and stomach, he was able to run home and inform his parents. (*Med. Gaz.*, vol. 3, p. 116.) This retention of muscular power cannot always be referred to the fact of the stomach containing food or liquid sufficient to dilute the poison, because, in Mr. Schwabe's case, the acid was not taken until some time after a meal. In a case which occurred to Dr. Walker the patient was able to get out of bed and sit on a night-stool, twenty hours after he had taken two ounces of oil of vitriol, and five hours before he died from its effects. These facts are important in a medico-legal view, as the following case will show. In December, 1843, a soldier was found lying on the pavement, and suffering from the effects of sulphuric acid. When questioned, he declared that he had been poisoned at a wine-merchant's shop. The man soon died, and the inspection showed that his death had been caused by sulphuric acid, taken probably in a diluted state. None of the acid was discovered in the matter last vomited, or in the stomach of the deceased; that which was *first* vomited had not been collected! Nevertheless, the cause of death was very clear. The wine-merchant's shop where the deceased said he had been poisoned was at some distance (not specified) from the spot where his body was found; and on the question being put to MM. Ollivier and Chevallier, they gave it as their opinion that the deceased could not have exerted a power of locomotion for so great a distance, and affirmed that, in their judgment, based upon cases fatal within a similar period of time, the deceased could have walked only a very short distance after swallowing the poison. They therefore inferred that it was a case of suicide, and not of homicide. (*Ann. d'Hyg.*, 1845, vol. 1, p. 179.) Considering the facts above detailed, and that the sulphuric acid was in this instance diluted, the medical opinion here given appears to have been somewhat stronger than prudence would warrant. A person who has taken sulphuric acid may undoubtedly retain a power of locomotion; but the degree to which it may be exerted must depend on the special circumstances of each case.

In October, 1856, W. V., æt. 56, swallowed by mistake a dessert-spoonful of oil of vitriol, and was admitted into Guy's Hospital. He was able to walk upstairs to his bed, and did not appear very ill, although dejected. The lining membrane of his mouth was of a brown color. He vomited slightly at first, and there was one fluid evacuation from the bowels of a brown color. On the two following days he appeared depressed, but there were no urgent symptoms. The case was considered slight, and there was every expectation that he would recover. He died suddenly on the fourth day. (*Guy's Hosp. Rep.*, 1859, p. 134.) Mr. Porter mentions the case of a girl, who after having swallowed a quantity of concentrated sulphuric acid, sat quietly down to tea with some friends, although the quantity of acid taken was sufficient to cause her death in a few hours. Another case is related in which a man took a second dose of the same acid, because he thought the first might not be sufficient. (*Med. Chir. Rev.*, vol. 28, p. 399.)

In general, it is observed that the symptoms continue to increase in severity until death, when the case is rapid, but there may be remissions, and, just before death, the pain and suffering have been observed to become considerably abated. In other cases, as in that above related, there may be an intermission of the symptoms, although the case may ultimately prove fatal.

Among the *secondary* symptoms of poisoning by this acid, when the person survives some days or weeks, should be mentioned profuse salivation. This was observed in Mr. Tatham's case. (*G. H. Rep.*, Oct. 1846, p. 396.) Salivation commonly occurs about the second or third day—sometimes later. Desgranges observed a miliary eruption on the skin among the secondary consequences of poisoning by sulphuric and nitric acids. (Belloc, *Cours de Méd. Lég.*, 120; Galtier, *Traité de Toxicologie*, vol. 1, p. 176.)

Appearances after Death.—Casper states from his observations, that the bodies of persons poisoned with sulphuric, and probably other mineral acids, resist putrefaction; they remain fresh for some time, and give out no offensive smell on inspection. He attributes this to the acid neutralizing the ammonia of the putrefactive process. (*Handb. der Ger. Med.*, vol. 1, p. 400, 429.) The effects produced by this acid are not always found in the stomach; they may be confined to the region of the throat and air-passages. In an inspection of the body, the whole course of the alimentary canal from the mouth downward, ought to be examined; since in all recent or acute cases, it is in the gullet and *throat* that we obtain strong evidence of the action of a corrosive poison. The discovery of the usual marks of corrosion in these parts is always highly corroborative of the signs of poisoning found in the stomach. During the inspection, the examiner must not omit to notice any spots on the skin produced by the action of the acid: these are commonly of a dark brown color, and are situated about the mouth, lips, and neck. The appearances met with in the body will vary according to whether death has taken place rapidly or slowly. Supposing the case to have proved rapidly fatal, the membrane lining the *mouth* may be found white, softened, and corroded; but in some cases this local change is not met with. It was just now observed that, when the acid has been administered by a spoon, the mouth may escape any chemical action. In the case of the *Queen* v. *Thomas* (Monmouth Lent Assizes, 1847) it was proved that the throat, gullet, and stomach of the deceased, an infant ten days old, were much corroded by sulphuric acid, which had been given to it in a diluted state; but there was no appearance of injury to the mouth. This was probably owing to a spoon having been used, and the poison having been poured down the throat slowly, as the mucous membrane was extensively corroded at the back part; and it was clear, therefore, that some corrosive substance had passed into the fauces. The mucous membrane of the throat and gullet will commonly be found corroded, having sometimes a brownish or ash-gray color. The corroded membrane of the gullet is occasionally disposed in longitudinal folds, portions of it being partly detached.

The *stomach*, if not perforated, is collapsed and contracted. On laying it open, the contents are commonly found of a dark brown color, and of a tarry consistency, being formed in great part of mucus and altered blood. The contents may or may not be acid, according to the time the patient has survived, and the treatment which has been adopted. On removing them the stomach may be seen traversed by black lines, or the whole of the mucous membrane may be corrugated, and of a dark brown or black color. This blackness is not removed by washing. On stretching the stomach, traces of inflammation may be found between the folds, indicated by a deep crimson-red color. On removing the blackened membrane, the red color indicative of inflammation may be also seen in the parts beneath. Both the dark color and marks of inflammation are sometimes partial, being confined to insulated portions of the mucous membrane. When the stomach is perforated, the coats are softened, and the edge of the aperture is commonly black and irregular. In one case the fore part of the stomach presented a number of small holes, having black margins. In removing the stomach, the aperture is apt to be made larger by the mere weight of the organ. The contents do not always escape; but when this occurs, the surrounding parts are attacked by the poison. In a case which occurred at Guy's Hospital, the spleen, the liver, and the coats of the aorta were found blackened and corroded by the acid, which had escaped through the perforation. The perforation of the stomach probably, in some instances, takes place after death from the chemical action of the acid. Dr. Craigie, of Edinburgh, thinks that even when there is no perforation of the stomach, the acid may find its way by transudation through the coats of the organ in a very short time after it has been swallowed. In a case in which two ounces of the strong acid had been taken, and the person died in three hours and a half, he found that the peritoneum and the fluid contained in it, reddened litmus-paper strongly. There was also a slightly acid reaction even in the serous membranes of the chest. It does not appear, however, that the nature of this acid was determined by the application of any test.

Dr. Hoffmann states as the result of his observation, that the corrosive action of sulphuric acid is greatest in an acid which contains 60 per cent. of the anhydrous compound, and that it is diminished in acids which are stronger or weaker. The longer the acid is in contact with the coats, the greater is the chemical action. The coats may be found thickened or softened; blood is effused, and this is converted by the acid into a black tarry liquid, characteristic of this form of poisoning. Sometimes the mucous membrane is hardened, and it may be removed in the form of an eschar. The surrounding parts are generally inflamed. (*Ann. d'Hygiène*, 1837, vol. 1, p. 231.) In the case of the infant E. B. (*ante*, p. 184) death took place in seven hours from a teaspoonful of oil of vitriol. The following appearances were found: The lips, tongue, pharynx, and the whole of the œsophagus were excoriated and of a brown color, the mucous membrane being destroyed in some parts. The top of the larynx was almost closed by an acute inflammatory œdema of the submucous tissue. The inner surface of the stomach presented a charred appearance, and was blackened throughout, the pyloric end being raised into a hard, black, massive layer. It contained a brownish fluid, but there was no sulphuric acid in it. The poison had not acted on the membrane beyond the pylorus. (*Guy's Hosp. Rep.*, 1857, p. 137.) The remarkable feature of this case is that so great an amount of local injury should have been caused by only one drachm of the acid.

When the person has survived for eighteen or twenty hours, traces of corrosion and inflammatory action may be found in the *small intestines*. In one instance the mucous membrane of the ileum was found corroded. In a case which occurred to Dr. Walker, of Inverness, a man died in twenty-five hours after he had swallowed nearly two ounces of oil of vitriol. On inspection, the mucous membrane of the stomach was destroyed, and the whole surface darkened. The greatest amount of injury was at the intestinal end, where three small perforations were found. The orifice of the pylorus was swollen, constricted, and hardened; it was so small as to admit only of a silver probe. The duodenum had also suffered much. The first two inches of the arch of the aorta were very much inflamed. (*Edin. Mon. Jour.*, June, 1850, p. 538.) Baron Dupuytren met with a case in which a woman died in seven hours from the effects of oil of vitriol. The mucous membrane of the gullet was raised by the action of the acid into longitudinal folds; that of the stomach was covered with black irregular spots; the coats were in a puffy state, and had a cauterized or burnt appearance. The pylorus and duodenum presented similar appearances. (*Med. Gaz.*, vol. 11, p. 813.) In the case of a boy poisoned by concentrated sulphuric acid, the inner lining of the œsophagus was puckered, dry, and brittle; it was readily detached from the parts beneath, and came off in small scale-like portions. The stomach was not perforated, the coats were thin, and allowed the contents to be seen through them. When opened, the whole of the mucous membrane was of a dark color, apparently stained by a bloody fluid, four ounces of which were contained in the stomach. The large end was unaltered, but the whole circumference of the smaller end, midway between the two openings, was black, irregular, rough, and thickened. The mucous membrane was here destroyed, blood had been effused, and this had been coagulated and darkened by the action of the acid. The blood adhered to the corroded membrane. (Roupell, *On the Effects of Poisons*, plate v.) The interior of the *larynx* as well as of the *bronchial tubes*, has also presented marks of the local action of the acid. The acid had thus destroyed life without reaching the stomach (*ante*, p. 185).

It is important for a medical witness to bear in mind that the throat and gullet are not always corroded; the mucous membrane sometimes presents merely black specks or points. Dr. Ogle met with a case in which the membrane covering the tongue was but slightly affected. The man had swallowed a large dose of the acid and had died in nine hours. (*Med. Times and Gaz.*, April 21, 1860.) Strange as it may appear, cases are recorded in which, notwithstanding the passage of the poison into the stomach, the gullet has escaped its chemical action. Mr. Dickinson has reported a case of poisoning with sulphuric aci is occasionally disposed in longitudinal folds, portions of it being partly detached.

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When the acid has been taken in a *diluted* state, the marks of inflammation on the mucous membrane are more apparent and the blackening is not so considerable. Nevertheless, the acid, unless too much diluted, acts upon and darkens the blood in the vessels, as well as that contained in the stomach, although is may not blacken the mucous membrane or the contents. Owing to the absence of corrosion in the throat and gullet, it might be assumed that sulphuric acid could not have been swallowed; and, in this respect, a case reported by M. Blondlot, of Nancy, is of some interest. This gentlemen was required to examine the clothes and viscera of an infant named Boullet, aged two months, that had died from the effects of sulphuric acid. The tongue, pharynx, and gullet presented no mark of corrosion, or of any appearance indicating that a corrosive substance had been in contact with them. There was no eschar or alteration of color in any part. The appearances in the stomach were not very striking. An analysis showed that sulphuric acid existed abundantly on the clothing, but not a trace of the poison could be detected in the viscera. The case was remitted to MM. Devergie, Barse, and Lesueur for examination; they confirmed the conclusions of M. Blondlot, and pronounced that notwithstanding the absence of marks of corrosion in the viscera, and of the acid from their contents, the child had died from sulphuric acid administered to it. They were inclined to attribute the absence of the poison to vomiting and elimination by the urine. (*Journal de Chimie Médicale*, 1846, vol. 2, p. 17.)

In the case of W. V., related at p. 186, in which the patient died on the fourth day, the appearances were peculiar. The mucous membrane of the mouth, tongue, epiglottis, and gullet was of a yellow color; the membrane could be easily peeled off. The parts about the larynx, where the acid had come in contact with them, were colored yellow and swollen. The mucous membrane of the œsophagus was only superficially colored. The substance of the gullet was inflamed and swollen to thrice its natural thickness. The stomach contained about a pint of bright yellow fluid, which was not acid, and which did not yield any sulphuric acid on analysis. The greater end of the stomach presented the same yellow color as the gullet. The pyloric, or intestinal half, was blackened, and raised into swollen masses or ridges. These would soon have sloughed off. The black appearance was owing to the chemical action of the acid on the blood effused in the submucous tissue. The coats of the stomach, although not perforated at this part, were readily torn. The dark appearance in the mucous membrane ended at the pylorus; but for the first three inches the folds (rugæ) of the duodenum (or small intestine) were slightly blackened. The remainder of the intestines presented no unnatural appearance. The small intestines contained a yellow fluid like that found in the stomach. The blood presented no remarkable appearance. The other organs were healthy. (*Guy's Hosp. Rep.*, 1859, p. 134.)

Chronic Poisoning.—The appearances just described will not, of course, be met with in protracted cases. If the person survive sufficiently long, all signs of inflammation and corrosion will disappear. Thus, in a case in which a child survived *twenty-five* days, the mucous membrane of the mouth and throat was sound but pale; that of the gullet, stomach, and duodenum was smooth, and equally free from any marks of corrosion or inflammation. (*G. H. Rep.*, Oct. 1846, p. 396.) Casper met with two cases in children; in one the child survived three days, and in the other eight days. In both, the mucous membrane was pale. In the case of three days' duration, there was no erosion of the gullet. (*Handb. der Ger. Med.*, vol. 1, p. 421.)

The subjoined case shows the appearances which were met with when death did not occur until the eleventh day: C. D., a female lunatic patient, æt. 55, was admitted into Guy's Hospital on Oct. 5, 1855, and died Oct. 16. Two hours before admission, and before her breakfast, she drank two ounces of a mixture of one part of sulphuric acid to four of water. On admission, no stains were perceived on the mouth—she was in a state of collapse—almost pulseless, the skin cold, and she was unable to swallow. In two hours the skin became warm, and the patient vomited some thick bloody liquid, which contained sulphuric acid. There was also a discharge of blood from the bowels. In about ten or twelve hours she was able to swallow milk and arrowroot. She continued to vomit and pass blood by the bowels for several days. She was much reduced in strength, but there were no very urgent symptoms. In four days after her admission, she was able to swallow without difficulty. She had purging, but without passing blood, and the vomiting ceased. On the day of her death she sat up and spoke as usual, but in the evening was unexpectedly found dead.

The body was inspected seventeen hours after death. The mucous membrane of the mouth was white, that of the throat and gullet was pale, and covered by a granular deposit (of epithelium). It was not corroded. The stomach was slightly contracted; it contained two ounces of a yellow fluid, like the yolk of egg, and a thin membrane of the same color attached by one end. This consisted of the lining membrane of the upper part of the stomach, traversed by vessels filled with coagulated blood. The central portion of the organ had also lost its mucous membrane. The destruction of the membrane continued into the duodenum. The small intestines were congested, and a false membrane was found in the jejunum. The large intestines were acutely inflamed, the interior being covered by adherent false membranes. The liver and kidneys were healthy. (*Guy's Hosp. Rep.*, 1859, p. 153.) In a case which proved fatal on the sixteenth day, the stomach was found perforated, but adherent to the coverings of the abdomen. The mouth, throat, and gullet presented no marks of corrosion. The quantity of acid taken (consisting of two-fifths of strong acid) amounted to two ounces. When first seen, the mucous membrane of the mouth was white. (*Med. Times and Gazette*, Dec. 19, 1857, p. 629.)

In other instances the mucous membrane has been found either entirely destroyed, or more or less ulcerated, and in some parts gelatinized. (*Med. Gaz.*, vol. 14, p. 31.) The stomach has been found much injected and swollen, and at the greater end there was softening and erosion. Obstinate constipation, with great difficulty in swallowing, were among the most urgent symptoms. (*Med. Gaz.*, vol. 17, p. 340.) This destruction of the inner coat of the stomach leads to death, by impairing the function of digestion. In several cases, the aperture of the pylorus has been found much contracted. (*Galtier*, op. cit., vol. 1, p. 197.) Sometimes stricture of the œsophagus is a consequence of the local action of the acid. (*Med. Times and Gaz.*, May 15, 1858, p. 510.) The common secondary causes of death in these chronic cases are fever, irritation, or exhaustion. There may be occasionally a difficulty in connecting death with the poison when the person survives for some weeks or months.

For a number of cases illustrative of the effects of this acid, see *Empoisonnement*, par Tardieu and Roussin, 1867, p. 203.

Fatal Dose.—The dangerous effects of sulphuric acid appear to arise more from its degree of concentration than from the absolute quantity taken. The quantity actually required to prove fatal must depend on many circumstances. If the stomach is full when the acid is swallowed, its action may be spent on the food and not on the stomach; and a larger quantity might thus be taken than would suffice to destroy life if the organ were empty. In one case, one drachm of sulphuric acid destroyed life in seven days; in another (Humphrey's case, *ante*, p. 84, also *Med. Gaz.*, vol. 8, p. 77), about one drachm and a half destroyed life in two days. In Mr. Schwabe's case six drachms destroyed life in twenty-four hours. (*Med. Gaz.*, vol. 36, p. 826.) In one instance, a patient survived fifty-five hours after taking three fluid ounces of the concentrated acid (Dr. Sinclair, *Med. Gaz.*, vol. 8, p. 624); in another, related by Sobernheim, a man swallowed an ounce and a half of the concentrated acid, and yet slowly recovered from its effects. (*Handbuch der Prakt. Tox.*, p. 684.) In a case, quoted by Dr. Craigie, a young woman, aged 18, recovered after having taken *two ounces* of concentrated sulphuric acid. She was completely restored in about eighteen days. (*Ed. Med. and Surg. Jour.*, April, 1840.) Another instance of recovery after two ounces of the concentrated acid had been taken, is reported by Mr. Orr (*Med. Gaz.*, vol. 3, p. 255). A remarkable instance of recovery from a large dose was observed in a case which occurred in the practice of M. Biett. The patient, a man, aged 31, swallowed by mistake *three ounces* (by weight?) of commercial sulphuric acid. Severe burning pain and vomiting immediately followed; the man fell and rolled on the ground in agony, but nevertheless was able to walk some distance to the hospital without assistance, although he rested occasionally. Milk and magnesia were freely given to him, and in a week he perfectly recovered. The most striking symptom was excessive salivation, which set in on the second day and continued for three days. (Galtier, *Toxicologie*, vol. 1, p. 186.)

It is probable that, in these instances of recovery from large doses, the greater part of the poison has been expelled in the matter first vomited. In a case which occurred to Dr. Letheby, the patient, a child only nine years old, recovered in a short time, after having swallowed one ounce of concentrated sulphuric acid. In this case nothing was done for five minutes; for the first few days the patient was copiously salivated. (*Med. Gaz.*, vol. 39, p. 116.) The smallest quantity which I have been able to meet with as having proved fatal, was in a case already quoted. Half a teaspoonful of concentrated sulphuric acid was given to a child, about a year old, by mistake for castor oil. The usual symptoms came on, with great disturbance of the breathing, and the child died in twenty-four hours. The quantity here taken could not have exceeded *forty drops*. It is, however, doubtful whether this small quantity would have proved fatal to an adult. The smallest fatal dose which Sir R. Christison states he has found recorded, was *one drachm;* it was taken by mistake, by a stout young man, and killed him in seven days. (*Op. cit.*, p. 162.)

Period at which Death takes place.—It has been already stated that the average period at which death takes place in cases of acute poisoning by sulphuric acid, is from eighteen to twenty-four hours. Death has frequently occurred suddenly and unexpectedly, when the patient appeared to be progressing to recovery. If the stomach is perforated by the acid, it proves more speedily fatal. In an instance, reported by Dr. Sinclair, in which a child about four years old died in four hours, the stomach was perforated. When the acid acts upon the air-passages, death may be a still more speedy consequence from suffocation; and, owing to this, it appears to be more rapidly fatal to children than adults. But in one case of an adult, elsewhere recorded (*ante*, p. 184), it must have destroyed life by its action on the air-passages during swallowing. Dr. Craigie mentions a case in which three ounces of concentrated sulphuric acid destroyed life in three hours and a half; but the shortest case on record is, perhaps, that mentioned by Remer in *Hufeland's Journal.* In this instance death took place in two hours. A case which proved fatal in two hours, is also reported by Casper. (*Handb. der Ger. Med.*, 1857, vol. 1, p. 422.) The stomach in this case was quite blackened, and so soft that it gave way like blotting-paper. The stomach was found perforated, and the omentum blackened. There had been some vomiting, but sulphuric acid was detected in the corroded parts. In the case of a child a year and a half old, criminally poisoned by its mother, death took place in *one hour.*

There were parchment-colored streaks and spots at the corners of the mouth, and on the arms and hands of the child. The stomach contained a dark acid fluid, consisting of blood and mucus; the coats were softened to solution. The gullet was firm, and the mucous membrane had a gray color. A case, in which life was destroyed with equal rapidity, is reported in the *Edinburgh Monthly Journal*, 1854 (p. 138). A woman took a wine-glassful of oil of vitriol (s. g. 1.833), in mistake for ale. In spite of treatment, she died in *one hour*. The stomach was perforated, and the acid had escaped; the coats were softened; the edges of the aperture were ragged; and the mucous surface, generally, was mottled with dark-brown patches. Another instance of death in two hours is quoted by Galtier (*Toxicologie*, vol. 1, p. 193). A case is reported by Mr. Watson, in which a woman swallowed two ounces of the strong acid. She died in *half an hour*, but it appears that a quarter of an hour before death she had made a deep wound in her throat, which caused a great loss of blood. The stomach was found extensively perforated; but it is highly probable that in this case the wound accelerated death. In a case which occurred to M. Rapp, a man, æt. 50, swallowed three ounces and a half of strong sulphuric acid, and died from the effects in three-quarters of an hour. (*Gazette Médicale*, Dec. 28, 1850.) Even when taken in the diluted state, it may destroy life rapidly. A man swallowed, on an empty stomach, six drachms of the strong acid diluted with eighteen drachms of water. He suffered from the usual symptoms, and died in two hours and a half. (*Med. Times and Gaz.*, 1868, vol. 1, p. 183.) Fifty-three deaths from sulphuric acid in England and Wales were recorded in a period of five years, 1863–7.

On the other hand, there are numerous instances reported in which the poison proved fatal from secondary causes, at periods varying from one week to many months. In one of these, a child recovered under treatment from the first effects, but died of starvation after twenty-five days, from the impossibility of retaining any kind of food on its stomach. (*G. H. Rep.*, Oct. 1846, p. 396.) A remarkable case of a similar kind occurred to Dr. Wilson, of the Middlesex Hospital, and is referred to by Mayo in his *Outlines of Pathology*. A young woman swallowed about a tablespoonful of sulphuric acid on January 4, and died from its effects on the gullet on November 14 following. She gradually wasted away, and sank from innutrition. This was forty-five weeks, or *eleven months*, after she had swallowed the poison. There is no doubt that the acid may prove fatal at all intermediate periods, and at intervals much longer than this; but the longer this event is protracted, the more difficult will it be to ascribe death to its effects. Dr. Beck refers to a case in which death took place from stricture of the œsophagus *two years* after the poison had been taken. (*Med. Jour.*, vol. 2, p. 426.)

Treatment.—Although it is the general practice to give magnesia and chalk freely in milk or water, it appears to me, from a case which I had the opportunity of examining, that a solution of carbonate of soda in milk and water, properly diluted, and given in small quantities at intervals, would act more effectually and more speedily in neutralizing the poison. The insoluble particles of calcined magnesia adhere closely to the mucous membrane, and do not readily come into contact with the acid. In examining the dark tarry matter vomited by a child half an hour after the concentrated acid had been taken, I found it still intensely acid, although, during the whole period, a magnesia mixture had been freely given in divided doses. This objection would not apply to the use of bicarbonate of magnesia or lime; and the evolution of carbonic acid would be a minor evil compared with the action of sulphuric acid in an unneutralized or imperfectly neutralized condition. Dinneford's fluid magnesia is well adapted to neutralize the acid poisons. It consists of bicarbonate of magnesia in a soluble form. Sobernheim and Simon relate several instances in which persons who had taken this poison, were apparently saved by the free use of these alkaline diluents. In the absence of these substances, a solution of soap in distilled or rain water, or even oil, may be freely administered.

There is often great difficulty in making the patient swallow, the throat being swollen and blocked up with shreds of tough coagulated mucus and phlegm. Hence it has been recommended to employ the stomach-pump for the purpose of injecting the liquids into the stomach. The use of this instrument ought, however, if possible, to be avoided; since it is very likely to lacerate and perforate the structures which may be softened and corroded by the acid. When there are symptoms of suffocation from an affection of the larynx, tracheotomy must be resorted to. On the whole, the antidotal treatment of cases of poisoning by sulphuric acid has not been very successful, the patient not having been seen sufficiently early by a medical man to give much hope of success. It should be remembered that the poison begins to act instantly; and if the stomach be at the time empty, there is but little prospect of saving the patient. These cases often prove fatal even when every trace of the poison has been removed from the stomach, owing to the extensive local injuries produced.

The following case of successful treatment, which was reported by Mr. Gardner to the *Lancet* (Aug. 25, 1838), deserves to be here mentioned. A young man swallowed half an ounce of strong sulphuric acid. The usual symptoms appeared; milk and carbonate of magnesia were freely given. This person recovered in twelve days. One of the secondary symptoms in this case was profuse salivation.

It is worthy of remark, that there have been several instances of recovery in which no chemical antidotes had been administered. The treatment consisted simply in giving large quantities of gruel and milk; and there is no doubt, that any thick viscid liquid of this description, as, for example, linseed oil and lime water, albumen, or flour and water, must be beneficial by mixing with the acid and arresting its corrosive effects. In short, such a liquid would act much in the same manner as the presence of a large quantity of food is known to act when the acid is swallowed soon after a meal. In all cases it would be advisable to combine the use of chemical antidotes with the copious administration of milk or mucilaginous drinks.

CHAPTER XXIV.

SULPHURIC ACID—CHEMICAL ANALYSIS IN THE SIMPLE STATE—FALLACIES IN TESTING—DETECTION IN LIQUIDS CONTAINING ORGANIC MATTER—DIALYSIS—ABSENCE OF THE POISON—EVIDENCE FROM DETECTION ON ARTICLES OF CLOTHING—QUANTITATIVE ANALYSIS—POISONING WITH SULPHATE OF INDIGO.

SULPHURIC ACID.

THIS acid may be met with either concentrated or diluted; and a medical jurist may have to examine it under three conditions: 1. In its simple state. 2. When mixed with organic matters, as with liquid articles of food or in the contents of the stomach. 3. On solid organic substances, as where the acid has been thrown or spilled on articles of dress or clothing.

In the Simple State.—If *concentrated* (oil of vitriol), the acid possesses these properties: 1. A slip of wood plunged into it is immediately carbonized or charred. 2. When boiled with wood, copper cuttings, or mercury, it evolves fumes of sulphurous acid; this is known by the odor, as well as by the acid vapor first rendering blue, and then bleaching starch-paper dipped in a solution of iodic acid. 3. When mixed with its volume of water great heat is evolved (nearly 200° F. in a cold vessel). In this diluted state the acid does not carbonize wood, and is not decomposed when boiled with copper cuttings.

The Diluted Acid.—For the acid in the *diluted* state, but one test need be applied: a solution of a salt of barium—the *nitrate* or *chloride of barium.* Having ascertained by test-paper that the liquid is acid, we add to a portion of it a few drops of nitric acid, and then a solution of barium salt. If sulphuric acid be present, a dense white precipitate of sulphate of barium will fall down: this is insoluble in all acids and alkalies. If this precipitate is collected, dried, and heated to full redness for a few minutes in a small platinum crucible (closely covered) with four or five parts of vegetable charcoal powder, it will, if a sulphate, be converted into sulphide of barium. In order to prove this: 1. A portion of the chemical mixture when cooled may be mixed with water, well stirred, and filtered. A pale yellowish liquid will be obtained, having an alkaline reaction, and giving a brown or black precipitate with a solution of acetate of lead. 2. If in small quantity, the mixture may be placed at once on glazed card (coated with carbonate of lead) and wetted, when a brown or black stain of sulphide of lead will be produced. 3. The powder may be heated in a tube with strong hydrochloric acid, when sulphuretted hydrogen will be copiously evolved, known by its smell and by its darkening a solution of a salt of lead when passed into it. If the quantity of precipitated sulphate is very small it may be mixed with one-third of its weight of cyanide of potassium and heated in a reduction-tube to full redness. This residue, placed on glazed card, wetted, or added to a solution of acetate of lead, gives the reaction indicative of the presence of a sulphide, proving that the original precipitate was a sulphate, and that sulphuric acid was present in the liquid submitted to analysis.

Diluted sulphuric acid does not carbonize organic substances which are immersed in it. The application of heat will only effect carbonization when the water of the acid is entirely evaporated. Thus, paper or linen, wetted with the diluted acid, becomes charred when dried and heated. This may serve as one method of identification in the absence of tests.

The delicate action of this test is such, that a solution containing not more than the $\frac{1}{25000}$th part by weight of sulphuric acid is precipitated by it. When the sulphuric acid is diffused through a minimum of water the barytic test gives a perceptible precipitate with the $\frac{1}{110}$th part of a grain of the acid. If, however, this small quantity be diluted with an ounce of water, the test produces no perceptible change. In these experiments distilled water must be used, since all kinds of river and spring water are precipitated by the test. With regard to the reduction of the precipitate to the state of sulphide by charcoal or cyanide of potassium, I have found that one-half grain of the sulphate of barium will yield satisfactory evidence; and a quarter of a grain will give traces of sulphur, although somewhat indistinct. This is equivalent to about one-eighth of a grain of common oil of vitriol (bihydrate). In cases of poisoning, however, we either find the acid in larger proportion, or it is altogether absent.

Objections to the Process.—Provided the process be carried out with pure materials, to the production of *sulphide* of barium, it is not open to any objection. There are some points, however, which require consideration: 1. A solution of alum, of any acid *sulphate*, or of *bisulphate of potash*, might be erroneously pronounced to be free sulphuric acid; for alum and the acid sulphates would, with the tests, give all the reactions which have been here described. The answer to this objection is very simple: We must slowly evaporate a portion of the suspected liquid in a platinum capsule, when there will be a saline residue if the solution contains alum, or any dissolved sulphate—otherwise not; for sulphuric acid should be entirely dissipated by heat. 2. The quantity of free sulphuric acid present might be erroneously estimated, in consequence of some simple medicinal sulphate (as Epsom salt) being mixed with it. This may be determined also by evaporation.

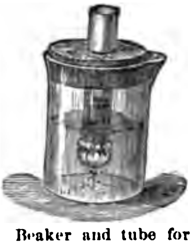
There is, however, another source of error: Any acid mixed with a common sulphate employed in medicine, might be mistaken for free sulphuric acid; as, for example, a mixture of lemon-juice or vinegar with sulphate of magnesia. This may be suspected when any saline residue is left on evaporating the mixture. In such a case it will be easy to procure by evaporation and incineration the sulphate from a given measure of the liquid, and we can then determine whether the sulphate of barium obtained is greater than or equal to the weight of alkaline sulphate present. With this precaution, it appears to me impossible that an analyst can mistake a solution of a neutral sulphate for a solution of sulphuric acid. (*Empoisonnement*, par MM. Tardieu et Roussin, 1867, p. 191.)

In Liquids containing Organic Matter.—If sulphuric acid be mixed with such liquids as ale, porter, coffee, tea, or milk, the process for its detection is substantially the same, the liquid being rendered clear by filtration previously to adding the test. The sulphate of barium, if mixed with organic matter, may be purified by boiling it in strong nitric acid; but this is not commonly necessary, as the reduction of the precipitate may be equally well performed with the impure as with the pure sulphate. Some liquids generally contain either sulphuric acid or a sulphate, such as vinegar, sherry wine, ale, and porter, but the acid is in small proportion; therefore, if there be an abundant precipitate, it is probable that free sulphuric acid is present in them. Should the liquid be thick and viscid like gruel, it may be diluted with water, and then boiled with the addition of a little acetic acid. For the action of the test, it is not necessary that the liquid should be absolutely clear, provided it be not so thick as to interfere mechanically with the subsidence of the precipitate. If the patient has been under treatment, the matters obtained from the stomach may have *no acid* reaction, owing to the copious administration of water and abundant vomiting, or from an antidote having been used, such as soda or magnesia. If, on adding the test to a *neutral* liquid, there is a precipitate, sulphuric acid can be present only in the shape of a *sulphate*. If this precipitate be abundant, it cannot be due to the presence of minute traces of sulphates in the gastric and salivary secretions; but still it would be improper to infer, from this chemical fact alone, that sulphuric acid had been swallowed, because it is well known that some saline sulphates, such as those of magnesia and soda, are often prescribed in large quantities medicinally, and it might be fairly objected to this evidence, that the precipitate was due to the presence of one of these salts. The symptoms and the appearances in the throat and stomach would here aid the witness in forming an opinion,—chemistry alone might mislead him.

A similar process may be applied to the examination of matters *vomited* and of the *contents of the stomach*, care being taken to separate the insoluble parts by filtration before adding the test. The *coats* of the stomach should be cut up and boiled in distilled water for some time, for the perfect extraction of the acid. The acid decoction filtered and concentrated by evaporation may then yield evidence of its presence.

When the acid is mixed with milk, decomposed blood and mucus, or other substances, rendering it thick and viscid, it may be separated by dialysis. A portion of the acid viscid liquid should be placed in a test-tube, about five inches long and one inch in diameter, open at both ends, the neck being securely covered with a layer of thin bladder. The tube is then immersed, mouth downwards, in a beaker containing distilled water. After some hours the acid will pass through the membrane, and may be detected in the water. This process may be employed as a trial test of the contents of the stomach when they have a strong acid reaction. In thus testing for sulphuric acid it must be remembered that a sulphate, like Epsom salt, may be present in the liquid, and an innocent acid like vinegar or lemon-juice

FIG. 5.



Beaker and tube for the dialysis of sulphuric acid.

may give the acid reaction equally as well as a precipitate with the barium test. To remove any fallacy on this ground, a portion of the liquid tested should be evaporated, and the residue incinerated, when the sulphate, if present, will be obtained.

It is a medico-legal fact of considerable importance, that the contents of a stomach in a case of poisoning by sulphuric acid, are sometimes entirely free from any traces of this poison, even when it has been swallowed in large quantity. The acid is not commonly found when the person has been under treatment, when there has been considerable vomiting, aided by the drinking of water or other simple liquids, or when he has survived several days. If the case has been under treatment, the acid is either wholly absent or neutralized by antidotes. A girl swallowed four or five ounces of diluted vitriol, and died in eighteen hours. No portion of the acid could be detected in her stomach; but she had vomited considerably, and the acid was easily proved to exist in the vomited matters, by examining a portion of the sheet of a bed which had become wetted by them. In another case, nearly two ounces of the concentrated acid were swallowed; the patient died in twenty-five hours; the stomach was extensively acted on, and yet no trace of the acid could be discovered in the contents. The liquidity of the poison, and the facility with which it becomes mixed with other liquids and ejected by vomiting, will readily furnish an explanation of this fact. In many cases of poisoning by sulphuric acid, therefore, a medical witness must be prepared to find that chemical analysis will furnish only negative results. This, however, is not inconsistent with death having taken place from the poison. The facts are so conclusive on this point, that I should not have thought it necessary to add to the evidence accumulated on the subject, but that an erroneous statement has been put prominently before the public to the effect that no person can die from poison, except the poison be found in the body. Casper has dealt with this question. He relates three cases of poisoning by sulphuric acid, which occurred to himself, one which proved fatal in eight days, a second in five days, and a third in three days. In not one instance could a trace of the poison be found. (*Handb. der Ger. Med.*, i, vol. 1, pp. 421, 429.) In the second case two tablespoonfuls were swallowed by a girl. The analysis revealed merely the accidental presence of a fractional part of a grain of alkaline sulphate in the stomach and bowels. Thus there was an entire failure of proof from chemistry, while the facts of the case, and the appearances in the body, established conclusively that death had really been caused by sulphuric acid. In one instance, in which death took place on the eleventh day, I found no trace of sulphuric acid in the body. If the stomach should be perforated, the contents will be found in the abdomen, or perhaps in the lower part of the cavity of the pelvis; they may then be collected, boiled with distilled water, and the solution examined for the acid by the process already described. If the contents of the stomach are highly putrefied, the sulphuric acid may be found combined with ammonia.

On Solid Organic Substances.—It sometimes happens in cases of poisoning by sulphuric acid that it is spilled upon articles of clothing, such as cloth or linen, or on the sheets of a bed, and here a medical jurist may succeed in detecting it, when every other source of chemical evidence fails. Again, sulphuric acid is often used for the purpose of seriously injuring a party, as by throwing it on the person, an offence which is treated as a felony, and renders the offender liable to a severe punishment. On such occasions, proof of the corrosive nature of the liquid is required; and this is easily obtained by a chemical examination of a part of the dress. A case of this kind was tried at the Liverpool Winter Assizes, 1866 (*Reg.* v. *Goff*). The injury appeared to be of a superficial kind. The jury found the prisoner guilty of throwing the corrosive fluid, but with no intent to injure. This was tantamount to an acquittal. A person committing this act with intent to injure is now guilty of a felony whether any bodily injury be done or not.

The process of analysis is very simple. The spot, unless it has been washed, strongly reddens litmus-paper when pressed upon it. The stained cloth should be digested in a small quantity of distilled water at a gentle heat, whereby a brownish-colored liquid may be obtained on filtration. If sulphuric acid is present, the liquid will have an acid reaction, and produce the usual effects with the barium test.

Strong sulphuric acid produces on black cloth stains which are brownish colored in the centre, becoming after a time of a dull red at the margin. The cloth is softened and remains damp from the absorption of moisture. It subsequently passes to the state of a black tarry substance in which the structure of the cloth cannot be recognized. Diluted sulphuric acid produces at once on black cloth a red stain which slowly becomes brown. Old stains are known by the complete destruction of the organic fibre; fresh stains by their dampness. The acid remains fixed in the stuff. I have thus detected sulphuric acid in clothing after the long period of twenty-seven years. The detection of spots of this acid on articles of dress, has in some cases served to supply the place of direct evidence from a chemical analysis of the stomach; and in other instances it has aided justice in fixing on an accused person the act of administration (*ante*, p. 85).

In all cases the analyst should examine an unstained portion of the stuff, whether woollen, linen, or cotton. Some articles of clothing yield an acid liquid to water, and I have thus detected sulphuric acid in dyed woollen socks. In a case tried in 1840 at the Central Criminal Court, the late Dr. R. D. Thomson found that the material of a stained hat gave traces of sulphuric acid even in the portion which was uninjured by the throwing of the acid. He attributed this to the use of alum and copperas in the black dye. By a comparative analysis, he found a larger proportion of sulphuric acid in a portion of the hat on which a part of the acid had fallen.

Quantitative Analysis.—It may be sometimes necessary to state how much sulphuric acid is present in a particular liquid. In order to determine this point, a portion of this liquid should be measured off, and the whole of the sulphuric acid present precipitated by a salt of barium. The sulphate obtained should be rendered pure by boiling it in nitric acid, then washed, dried, and weighed. For every one hundred grains of dried sulphate obtained, we must allow half the weight, *i. e.*, fifty grains of common oil of vitriol (bihydrate) to have been present; hence the rule is a very simple one. As the equivalent of the bihydrated acid is 58, and that of sulphate of barium 116, the proportion of liquid acid is always equal to one-half of the weight of the precipitate. A teaspoonful or one fluid drachm of common oil of vitriol weighs 119 grains. An ounce of any organic liquid containing this quantity of the acid would therefore yield a precipitate of sulphate of barium weighing 238 grains.

SULPHATE OF INDIGO.

This is a dark-blue liquid, consisting of one part of indigo dissolved in nine or ten parts of oil of vitriol. It is much used in dyeing, and has given rise to some accidents. The symptoms and appearances are similar to those which have been described as produced by sulphuric acid. This kind of poisoning may be suspected, when, with these symptoms, the membrane of the mouth has a blue-black color. The vomited matters, as well as the fæces, are at first of a deep-blue tint; afterwards green; and it was observed in two instances that the urine had a blue tinge.

Symptoms.—One of the cases, reported by Orfila, was that of a child, who died in seven and a half hours. The other was observed by M. Bouchardat, and is of some interest. A young woman, aged 18, swallowed—as it was conjectured—about an ounce of the sulphate of indigo. Immediately afterwards, she felt an acute burning pain in the throat and in the stomach. She threw herself on the ground, and her cries soon brought around her her neighbors, who found her vomiting a bluish-colored liquid, which effervesced on the pavement. A quantity of oil and milk was immediately given to her; the milk was speedily thrown up coagulated, and of a blue color. When brought to the hospital, three hours afterwards, she was in the following condition: her face pale, features somewhat altered; her eyes were sunk, and her lips of a violet tinge. There was a yellowish-colored spot on the upper lip, at each angle of the mouth. The tongue was blue, the throat was painful, and there was a sense of constriction. The region of the stomach was tender. There was no pain in the abdomen; obstinate constipation; respiration difficult; great anxiety; coldness of the upper extremities; and a quick and small pulse. Her intellect was clear, and her answers to the questions put were sensible and proper. Four drachms of calcined magnesia were administered in a pint of water; much of this was rejected by vomiting, accompanied by bluish clots. A few hours afterwards the pain in the throat was very severe; the upper extremities were cold, and the pulse was imperceptible. The urine which she passed had a slight tinge of blue. She continued to become worse; the vomiting of chocolate-colored matter returned; and she died in about eleven hours after having taken the poison. Only one case of poisoning by this compound was brought into Guy's Hospital in fourteen years, 1860 to 1874. The patient recovered.

Appearances.—The body was examined twenty-seven hours after death. The head presented no particular appearance. There was no sign of corrosion in the mouth. The mucous membrane of the throat and gullet was easily detached in dry, white, brittle layers. The heart was filled with three ounces of coagulated blood; the aorta was also filled with brown and semi-liquid clots; the lining membrane of this vessel was of a bright-red color! The stomach was distended, and contained two ounces of a brown-colored liquid. The mucous membrane was carbonized, and of the color of soot, with slight patches of redness throughout its whole extent, except for about an inch near the pylorus, where it was of a rose-red color. It was easily detached in layers, but there was no trace of ulceration. The membrane of the duodenum was inflamed and ulcerated, and in parts it was found corroded and blackened. A dark-colored mucus was seen in the small intestines, and patches of a blue color were scattered through the colon. The femoral arteries were filled with a semi-coagulated dark-colored blood. The cavity of the left femoral artery was completely obstructed by a clot. M. Bouchardat, who reports this case, considers that the deceased died from the absorption of the acid into the bloodvessels, by which the fibrin of the blood was coagulated, embolism produced, and the circulation arrested. Several instances of recovery are on record. Dr. Galtier reports two—one of which is the case of a young woman, who swallowed rather more than an ounce of sulphate of indigo. She recovered on the eighth day. Calcined magnesia and milk were found to be the best remedies (*Toxicologie*, vol. 1, p. 206), but fluid magnesia would be more efficient.

Analysis.—The process is the same as that described for sulphuric acid in organic mixtures (*ante*, p. 197). The blue color of the sulphate is immediately destroyed by boiling it with nitric acid. The barytic test may then be employed in the usual way.

CHAPTER XXV.

POISONING BY NITRIC ACID OR AQUA FORTIS—ACTION OF THE CONCENTRATED AND DILUTED ACID—NOXIOUS EFFECTS OF THE VAPOR—APPEARANCES AFTER DEATH—CHRONIC POISONING—QUANTITY REQUIRED TO DESTROY LIFE—PERIOD AT WHICH DEATH TAKES PLACE—PROCESSES FOR DETECTING THE POISON IN PURE AND ORGANIC LIQUIDS—DIALYSIS OF THE ACID—DETECTION ON ARTICLES OF CLOTHING.

NITRIC ACID is popularly known under the name of *aqua fortis*, or red spirit of nitre. According to Tartra, it seems to have been first used as a poison about the middle of the fifteenth century. Although it is perhaps much more used in the arts than oil of vitriol, cases of poisoning by it are by no means so common. Tartra was only able to collect fifty-six cases, extending over a period of nearly four hundred years. (*Traité de l'Empoisonnement*, 1802.) It appears from the return of inquisitions for 1837–8, there were only two fatal cases reported to have occurred in England during those years. In the more recent return, for 1863, the number of fatal cases amounted to 16. Two only were received into Guy's Hospital in a period of fourteen years, 1860 to 1874, of which one proved fatal. Cases of poisoning by this acid have been chiefly the result of accident or suicide. I have only met with one instance where it was poured down the throat of a child for the purpose of murder. The *external* application of nitric acid has been a criminal cause of death on several occasions—in one case the acid was poured into the ear of a person while sleeping, and it led to the slow destruction of life. These are not strictly cases of poisoning, but more nearly approximate to death from wounding or mechanical violence.

Symptoms—The Concentrated Acid.—The symptoms, on the whole, are similar to those produced by sulphuric acid. They come on *immediately*, and the swallowing of the acid is accompanied by intense burning pain in the throat and gullet, extending downwards to the stomach. There are gaseous fumes and eructations, more copious than in poisoning by sulphuric acid, from the chemical action of the poison, with swelling of the abdomen, violent vomiting of liquid or solid matters, mixed with altered blood of a dark brown color, and shreds of mucus and membranous flakes, of a yellow color, having a strong acid reaction and a peculiar odor. The abdomen is generally tender; but in one well-marked case of poisoning by this acid, the pain was chiefly confined to the throat; probably the poison had not reached the stomach. The mucous membrane of the mouth is commonly soft and white, after a time becoming yellow, or even brown; the teeth are also white, and the enamel is partially destroyed by the chemical action of the acid. There is great difficulty of speaking, as well as of swallowing, the mouth being filled with viscid mucus; the power of swallowing is sometimes entirely lost. Marks on the skin, from the spilling of the acid, are at first whitish, then yellow, and afterwards brown. On opening the mouth, the tongue may be found swollen and of a citron color; the tonsils are also swollen and enlarged. The difficulty of breathing is occasionally such as to render tracheotomy necessary, especially in young persons. (Case by Mr. Arnott, *Med. Gaz.*, vol. 12, p. 220 and p. 207, *post.*) As the symptoms progress, the pulse becomes small, frequent, and irregular; the surface of the body cold, and there are frequent rigors. The administration of remedies, even the swallowing of the smallest quantity of liquid, increases the severity of the pain, occasions vomiting, and gives rise to a feeling of laceration or corrosion. (*Tartra*, 144.) There is obstinate constipation, with, occasionally, suppression of urine. Death takes place in from eighteen to twenty-four hours, and is sometimes preceded by a kind of stupor, from which the patient is easily roused. The intellectual faculties, however, commonly remain clear until the last. In one instance, the patient was insensible, but she ultimately recovered.

Death may be occasioned by this acid, in consequence of its action on the larynx, as in the case of sulphuric acid. Should the patient survive the first effects of the poison, the mucous membrane of the throat and gullet may be ejected, either in irregular masses, or in the form of a complete cylinder of the œsophageal lining. There is great irritability of the stomach, with frequent vomiting and destruction of the powers of digestion; the patient becomes slowly emaciated and dies from starvation or from exhaustion. A man swallowed nitric acid in beer; he recovered from the first symptoms, but died six months afterwards, evidently from the injury caused by the poison to the mucous lining of the stomach. He suffered from pain and from such irritability in this organ, that neither solids nor fluids could be retained. (*Lancet*, Nov. 24, 1860, p. 510.) In a case which proved fatal in St. Bartholomew's Hospital, in March, 1851, the following symptoms were observed. The man took an ounce of strong nitric acid. He immediately vomited, but did not suffer much pain. When brought to the hospital, about half an hour afterwards, he looked pale and haggard, the skin was cold, and the pulse very feeble. The vomiting had quite ceased. He complained of no pain in the stomach, but of some uneasiness about the throat. He lay quiet for several hours, occasionally drinking a mixture of magnesia and water; he then became restless, and complained of severe pain in the abdomen, which increased in severity. He suffered greatly for three or four hours, and then died, having survived the taking of the poison about fifteen hours.

R. G., æt. 26, was admitted into Guy's Hospital in March, 1857. On the previous day he had drunk about a teaspoonful of nitric acid in mistake for vinegar. Immediately his lips, tongue, and throat began to burn. On admission his countenance was anxious, and pulse quick. His lips and tongue had upon them a thick brown scab; his mouth was very tender, and he had a constant burning sensation in it. Glycerin was applied. On the next day he complained of pain in the stomach. The scabs on the lips were beginning to come off, and the mouth and tongue had a bright yellow color. He continued to improve, and left the hospital in a week. (*Guy's Hosp. Rep.*, 1859, p. 140.)

Vapor of the Acid.—The *vapor* of this acid may destroy life, by its action on the lungs. In March, 1854, Mr. Haywood, a chemist of Sheffield, lost his life under the following circumstances: He was pouring a mixture of nitric and sulphuric acids from a carboy containing about sixty pounds, when by some accident the vessel was broken. For a few minutes he inhaled the fumes of the mixed acids, but it does not appear that any of the liquid fell over him. Three hours after the accident, he was sitting up and appeared to be in moderately good health. He was then seen by a medical man, and complained merely of some cuts about his hands. He coughed violently. In three hours more there was difficulty of breathing, with increase of the cough. There was a sense of tightness at the lower part of the throat, and the pulse was hard. At times he said he could scarcely breathe. He died eleven hours after the accident. On inspection, there was congestion of the trachea and bronchial tubes, with effusion of blood into the latter. The heart was flaccid, and contained but little blood; and the lining membrane of the heart and aorta was slightly inflamed. The blood gave a slightly acid reaction with test-paper. The larynx was not examined. It is very probable that the seat of mischief was in this organ, and that the deceased had died from inflammatory effusion and swelling of the parts about the opening of the windpipe. (*Lancet*, April 15, 1854, p. 430.) A similar accident occurred to Mr. Stewart and one of the janitors of an educational institution in Edinburgh, in March, 1863. A jar of nitric acid, which he was carrying, fell on the floor and was broken. He and the janitor, instead of withdrawing from the spot, wiped the floor, and attempted to save some of the acid. They thus inhaled the fumes which were immediately diffused. Mr. Stewart returned home unconscious of the mischief which had been done. After an hour or two, difficulty of breathing came on, and, in spite of every medical effort to save his life, he died in ten hours after the accident. The janitor suffered from similar symptoms, and died the day following. (*Chemical News*, March 14, 1863, p. 132.) It is probable that in these cases there was great bronchial effusion, leading to the entire obstruction of respiration. The fumes of nitrous acid vapor, which is generally associated with nitric acid, are of a very deadly kind. In the manufacture of gun-cotton these acid vapors are evolved, which, if respired, although they may produce no immediate ill effects, are liable to cause pneumonia and death. On one occasion, in preparing gun-cotton, I accidentally inhaled the vapor, and suffered from severe constriction of the throat, tightness in the chest, and cough for more than a week. M. Tardieu has published a report of the cases of two workmen, who lost their lives by breathing the nitrous fumes of a sulphuric acid chamber. They had entered it for the purpose of cleaning it. Two others, who accompanied them, suffered severely from the vapor, but recovered. (*Empoisonnement*, 1867, p. 219.)

The Diluted Acid.—The symptoms above described apply to *acute* cases of poisoning by concentrated nitric acid. When the acid is *diluted*, they are somewhat modified according to the degree of dilution. A remarkably instructive case of poisoning with diluted nitric acid has been published by Dr. Puchelt, of Heidelberg;—it shows not only the progress of the symptoms, but also the powers of nature in resisting for a time the chemical destruction of an important organ. A man, aged 52, swallowed two ounces of diluted nitric acid (the strength not stated). He was *immediately* seized with severe burning pain in the mouth and throat; and this was followed by vomiting, whereby the greater part of the acid was probably ejected. He was not seen for several hours, and then the symptoms had so far subsided that the hospital assistant sent him away as not requiring immediate attendance. An oily emulsion was subsequently given to him. After the lapse of thirty-six hours, he was admitted into the hospital, and was for the first time seen by Dr. Puchelt. The mucous lining of the mouth and pharynx was covered with a white shreddy membrane, which could be readily peeled off; parts were already abraded. There were yellow stains on the cuticle around the mouth, especially upon the upper lip. The patient experienced great difficulty in swallowing; the breathing was laborious, the stomach tender, and the abdomen was hard and retracted. On the whole, the symptoms were very favorable, and led to the suspicion that but little injury had been done to the stomach. Leeches and other antiphlogistic means were employed, and in about eight days he began to retain a portion of food on the stomach. Nevertheless, his strength diminished, and he became emaciated: on the fifteenth day the food which he took was rejected; on the sixteenth, some blood was found mixed with the stools; on the seventeenth, there was great pain, with vomiting of black fluid blood, and of decomposed membrane of a fibrous structure, which, when spread out, was a foot in breadth. This membrane was marked with black spots, as if it were burnt, and perforated with numerous small and large apertures. A large quantity of black putrid blood was at the same time passed by stool. The symptoms became after this more unfavorable, and the vomiting of blood frequently recurred, until death took place on the *twenty-third day* after the poison had been swallowed.

There is no doubt that the diluted acid was in this case much stronger than that of the British Pharmacopœia, which contains 14.95 per cent. of anhydrous nitric acid; or three parts of nitric acid to seventeen parts of water, its specific gravity being 1.101. I have not met with any instance of poisoning with this diluted acid.

Appearances after Death.—A full account of these will be found in the well-known work of Tartra, *Essai sur l'Empoisonnement par l'Acide Nitrique*, published An x. Supposing death to have taken place rapidly, the following appearances may be met with. The skin of the mouth and lips will present various shades of color, from an orange-yellow to a brown; it appears like the skin after a blister or burn, and is easily detached from the subjacent parts. Spots, produced by the spilling of the acid on the skin, may be found about the hands and neck. These are at first yellow, but when dry, they assume a brownish color. The yellow color is heightened by alkalies. A yellow frothy liquid escapes from the nose and mouth, and the abdomen is often much distended. The membrane lining the mouth is sometimes white, at others of a citron color; the teeth are white, but present a yellowish color at their junction with the gums. The fauces and larynx are much inflamed; the latter sometimes swollen. The lining membrane of the gullet is softened, and of a yellow or brown color, injected (contains more blood), is easily detached, often in long folds. The windpipe is more vascular than usual, and the lungs are congested. The most strongly marked changes are, however, seen in the stomach. When not perforated, this organ may be found distended with gas, its mucous membrane partially inflamed with patches of a yellow, brown, or green color, or it may be even black. This green color is due to the action of the acid on the coloring matter of the bile; but it must be remembered that a morbid state of the bile itself often gives this appearance to the mucous membrane in many cases of death from natural disease. There is occasionally inflammation of the peritoneum, and the stomach has been found glued to the surrounding organs. Its coats are often so much softened, as to break down under the slightest pressure. In the duodenum similar changes exist; but in some cases the small intestines have presented no other appearance than that of slight redness. It might be supposed that the stomach would be in general perforated by this very corrosive substance; but this is far from being the case. Tartra only met with two instances, and in one of these, the person survived twenty, and in the other thirty hours. In giving this poison to rabbits, I have not found the stomach perforated, although the acid had evidently reached this organ, from its coats being stained of a deep yellow color. In these experiments the non-perforation appeared to be due to the protective influence of the food with which the stomach was distended.

In the few cases that are reported in English journals, the stomach has not been commonly perforated; the poison was swallowed soon after a meal, and its coats had thus escaped the corrosive action of the acid. In the case which terminated fatally after the long period of six months there was, at the intestinal end of the stomach, a distinct cicatrix with puckering and hardening of the surrounding mucous membrane, causing a slight contraction of the pyloric orifice. The only other appearance consisted in some dark longitudinal lines on the posterior surface of the lining membrane of the gullet. This had probably been caused by the acid. (*Lancet*, Nov. 24, 1860, p. 510.) In the case which proved fatal at St. Bartholomew's Hospital (*ante*, p. 204) the stomach was extensively destroyed; the surface was not stained yellow, but the mucous membrane was removed by corrosion, and the coats beneath were partly reddened and partly blackened, as a result of the action of the acid on the blood in the vessels. In a case which occurred at the Hôtel Dieu at Lyons, the stomach was distended with gas and perforated at the greater end, the opening being partially plugged by the spleen, which had become adherent over it. In the small intestines there were numerous sloughs. In a case of poisoning by this acid which occurred at Guy's Hospital in July, 1871, Dr. Stevenson thus describes the appearances presented by the stomach: The man, æt. 21, had swallowed three fluid ounces of the commercial acid, and died in seventeen hours afterwards. The lips and angles of the mouth, as well as the forepart of the tongue, were yellow; but beyond this, from the œsophagus to the stomach, the mucous surface presented a milk-white opacity. At the lower part of the œsophagus, the mucous membrane was partly removed. The mucous membrane of the stomach was covered with a reddish-brown gritty paste, neutral to litmus. On removing this, the membrane was paler red towards the pylorus, and covered with numerous close-set ulcers. At the greater end, the mucous and submucous coats were destroyed. The rugæ had suffered most extensively. On the anterior surface of the stomach, near the lower border, there was a small hole, and at several other points the walls of the stomach were nearly perforated, the serous membrane above them being discolored. When floated on water, the greater part of the mucous surface was found to be flocculent with shreds of broken-down membrane. The duodenum showed sloughing of the valvulæ conniventes. The jejunum and ileum were natural. (*Guy's Hosp. Rep.*, 1872, p. 223.)

In Mr. Arnott's case (p. 203), a boy, æt. 13, supposing that he was going to drink beer, swallowed a mouthful of a fluid which proved to be nitric acid. Acute pain was felt in the mouth and throat. Magnesia was administered, and vomiting was quickly induced. The vomited matters consisted of a large quantity of food partly digested. There was great constitutional depression, but the chief distress was from symptoms indicative of inflammation of the larynx. Mr. Arnott performed the operation of opening the larynx with some relief to the boy, but he died in thirty-six hours from the time of swallowing the acid. On inspection sixteen hours after death, the effects of the acid were found to be confined to the tongue, palate, fauces, tonsils, and lining membrane of the throat and gullet. None of the acid had entered the larynx, but there was a layer of coagulated lymph on the mucous surface of the windpipe, arising from inflammation which had extended from the parts adjacent. The base, edges, and tip of the tongue, with the lower part of the gullet, were deprived of their investing membrane. The portion of membrane which remained adherent had a citron color. That which covered the tongue was ragged at its edges, that of the throat and gullet was dry, corrugated, and marked with longitudinal and transverse lines. It could everywhere be readily stripped off, the part beneath appearing red. The edges of the glottis were swollen; the epiglottis was destroyed. There was no trace of the effects of the acid in the stomach except at the lesser end, where the orifices of the mucous glands presented a citron color, like that of the throat. The mucous membrane of the stomach was probably protected from the action of the acid by the quantity of food contained in the organ. (Roupell, on the *Effects of Poisons*, pl. 4; *Med. Gaz.*, vol. 12, p. 220, and vol. 14, p. 489.)

In cases of *chronic* poisoning, *i. e.*, where death takes place *slowly*, the appearances are of course different, as the following case will show. A man, aged 34, swallowed a wineglassful of nitric acid, but the greater portion was immediately rejected by vomiting. An attack of acute gastritis followed, which was combated by the usual remedies. The man was discharged from the hospital into which he had been admitted in three weeks; but about a month afterwards he was readmitted, in consequence of his suffering from severe pain extending down the gullet to the stomach, as well as from vomiting after taking food. The patient gradually sank, and died *three months* after he had taken the acid. On dissection the pylorus was found so diminished in size, that its diameter did not exceed a line or two, and the duodenum was equally contracted for about an inch and a half from its commencement. The mucous membrane was softened and red in patches, and there were several cicatrices of ulcers. The subjacent tissues were in a scirrhous state. (See *Med. Chir. Rev.*, vol. 28, p. 553.)

In Dr. Puchelt's case (*ante*, p. 205), in which death took place on the twenty-third day, when the abdomen was opened there was no appearance of a stomach, but in its place a cavity formed by the liver, colon, and other viscera; the interior wall, lesser curvature, and upper part of the posterior wall, being wholly absent. A dark-green mass was spread over the interior; but the parietes were so soft as to give way on the slightest pressure. The intestinal canal, with the exception that it contained a large quantity of bloody matter, presented nothing peculiar. The mucous membrane of the gullet was removed throughout its whole length. (*Ein Fall von Vergiftung mit Scheidewasser*, von Dr. F. A. B. Puchelt, Heidelberg, 1845.) As in chronic poisoning with sulphuric acid the pylorus may be found much contracted. One instance of this has been given above; another occurred to Dr. Vernois, and is quoted by M. Tardieu (*Empoisonnement*, 1867, p. 234). The man had suffered for several years from symptoms of chronic gastritis which had followed the ingestion of a certain quantity of nitric acid. The symptoms assumed an acute form, and the man died. There was a thickening of the stomach near the pylorus, and the orifice was so reduced in size that it was barely possible to introduce a probe.

Fatal Dose.—The remarks made on this subject, in speaking of sulphuric acid, apply here. Tartra states that the quantity usually taken, in the cases which he collected, varied from one or two drachms to four ounces. Indeed, the obtaining of any information of this kind is purely accidental; and the determination of the exact quantity swallowed must be therefore very difficult. One point is certain;—a similar dose will not kill two persons in the same time—one may die slowly, and the other rapidly, according to whether the stomach at the time contains food or not. The *smallest* quantity which I find reported to have destroyed life is about *two drachms.* It was in the case of a boy, aged 13; he died in about thirty-six hours. In a case which occurred to Dr. Warren, a woman died from a similar dose in fourteen days. But less than this, even one drachm, would doubtless suffice to kill a child; and, under certain circumstances, an adult; for the fatal result depends on the extent of the mischief produced by this corrosive poison in the throat, windpipe, and stomach.

What is the largest dose of concentrated acid from the effects of which a person has recovered it is difficult to say; since in most cases of recovery mentioned by authors the quantity of the poison actually swallowed was unknown. In one instance a woman, æt. 26, recovered in a few days after having swallowed *half an ounce* of aqua fortis of the usual strength. There was great reason to believe, either that the poison did not reach the stomach, or that it produced but little action on this organ. The chief seat of pain was in the throat and gullet. (*Lancet*, May 8, 1847, p. 489.) Another case of recovery from about half an ounce of the strong acid mixed with the diluted acid is reported in the *Lancet* (1870, vol. 1, p. 549). The patient was a man, æt. 21. He had the usual symptoms, with the exception that there was no yellowness of the teeth nor corrosion of the mouth. The vomited matters were bloody and of a dark color. He suffered from stricture of the œsophagus, and this remained when he left the hospital about fifteen weeks after his admission.

Period at which Death takes place.—This must depend on the quantity swallowed, the strength of the acid, and whether any medical treatment has or has not been adopted. Out of twenty-seven deaths from nitric acid, reported by Tartra, in nineteen it destroyed life rapidly, and in eight slowly. This author met with two instances in which death took place within *six* hours after the poison was swallowed; but he considers that the greater number who fall victims to the direct effects of the acid die within twenty-four hours. Sobernheim relates a case of poisoning by nitric acid, which proved fatal in *one hour and three-quarters.* (*Op. cit.*, p. 402.) This I believe to be the most rapidly fatal case on record, where the acid has acted through the stomach. The usual well-marked effects were found in the gullet, stomach, and duodenum. In infants, however, life may be destroyed by this poison in a few minutes, should it happen to affect the air-passages. A woman, shortly after her delivery, in the absence of her attendants, poured a quantity of nitric acid into the mouth of her infant. She concealed from those about her this attempt at murder; but medical assistance was immediately sent for. The child died in a *few minutes*. Some of the acid had been spilled; and from the yellow color of the stains the medical man suspected that the child had been poisoned by aqua fortis. On inspection, nitric acid was found in its stomach, and the mother confessed the crime. (Cazauvieilh, *Du Suicide et de l'Aliénation Mentale*, p. 274.) Although in the report of this case the condition of the throat and larynx is not stated, it is highly probable, from the rapidity with which death took place, that this event was in great part due to suffocation.

With regard to the *longest* period at which death has taken place from the effects of this poison, a case has been already related, where a man who had swallowed nearly two ounces, did not die until three months afterwards (*ante*, p. 208). A case is recorded by Tartra, in which a woman died from exhaustion, produced by the secondary effects of the poison, eight months after having swallowed the acid. The most protracted case which I have met with is reported by M. Tardieu. (*Op. cit.*, p. 220.) It occurred to M. Moutard Martin. A woman, æt. 30, survived the effects of this acid for the long period of two years. She had died from starvation, and after death, the œsophagus was found contracted through its whole extent. There was a general thickening of the tube, and in some points it was more contracted than in others. The contraction was greatest at the lower part. (*Op. cit.*, p. 234.)

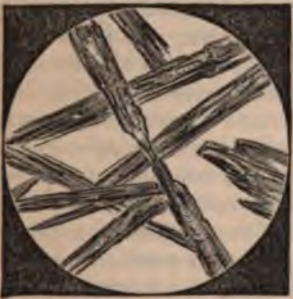
Treatment.—It may be the same as that recommended in poisoning by sulphuric acid. In addition to the remedies there suggested, a diluted solution of carbonate of soda, or fluid magnesia, with barley-water, and other demulcents, may be administered. In many cases there is an utter impossibility of swallowing even the smallest quantities of liquid; and if an attempt be made to introduce these remedies by a tube, there is great risk of perforating the softened parietes of the pharynx, larynx, or gullet. Should suffocation be threatened, tracheotomy may be resorted to. Modern experience is rather adverse to the recovery of these cases under any form of treatment; but according to Tartra, in accidental poisoning by this acid, there is great hope of recovery, if the patient receives timely assistance. He states that out of thirty-one cases, twenty-three recovered, seventeen perfectly; while out of twenty-four cases, wherein suicide was attempted, only six recovered. (*Op. cit.*, p. 186.)

CHEMICAL ANALYSIS.

Nitric acid may be met with either concentrated or diluted. The *concentrated acid* varies in color from a deep orange-red to a light straw-yellow. The sp. gr. of a sample of the acid was 1.392. A teaspoonful was equivalent to 79 grains, and a tablespoonful (half an ounce) to 316 grains. It may be recognized, 1. By evolving acid fumes when exposed. 2. By its staining organic matter yellow or brown, the color being heightened and turned to a reddish tint by contact with caustic alkalies. 3. When mixed in the cold with a few copper filings, it is rapidly decomposed—a deep red acid vapor is given off, and a greenish-colored solution of nitrate of copper is formed. Tin or mercury may be substituted for copper in this experiment. 4. It does not dissolve gold-leaf even on boiling; but on adding to the boiling liquid a few drops of hydrochloric acid, the gold is immediately dissolved.

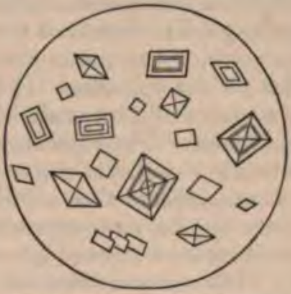
In the *diluted* state. This acid is not precipitated like the sulphuric by any common reagent, since all its alkaline combinations are soluble in water—1. The diluted liquid has a highly acid reaction, and on boiling it with some copper turnings, red fumes of nitrous acid vapor are given off, unless the proportion of water is too great. At the same time, the liquid acquires a blue color. 2. A streak made on white paper with the diluted acid does not carbonize it when heated; but a scarcely visible yellow stain is left. Diluted sulphuric and hydrochloric acids carbonize paper under similar circumstances. 3. The liquid is neither precipitated by a salt of baryta nor by nitrate of silver. These two last experiments give merely negative results—they serve to show that the sulphuric and hydrochloric acids are absent.

FIG. 6.



Crystals of nitrate of potash, magnified 30 diameters.

FIG. 7.



Crystals of nitrate of soda, magnified 30 diameters.

A portion of the acid liquid should now be carefully neutralized with potash, and then evaporated slowly to obtain crystals. If the liquid contained nitric acid, these crystals will possess the following characters: 1. They appear in the form of lengthened fluted prisms, which neither effloresce nor deliquesce on exposure. One drop of the solution evaporated spontaneously on glass will suffice to yield distinct and well-formed crystals. (Fig. 6.) This character distinguishes the *nitrate* of potash from a large number of salts. When neutralized with a solution of soda, the crystals are of a rhombic form—a very striking (microscopic) character of *nitrate* of soda. (Fig. 7.) 2. When moistened with strong sulphuric acid, the powdered crystals slowly evolve a *colorless* acid vapor. By this test the nitrate is known from every other deflagrating salt. 3. A portion of the powdered crystals should be placed in a test-tube and mixed with their bulk of fine *copper* filings.

The mass is then to be moistened with water, and a few drops of strong *sulphuric acid* added. Either with or without the application of a gentle heat, a decomposition ensues, by which red fumes of *nitrous acid* are evolved, recognizable by their color, odor, and acid reaction. If a tube only one-eighth of an inch in the bore, be used for this experiment, one-tenth of a grain of nitre will give satisfactory results. This is equivalent to about one-twentieth of a grain of nitric acid—a quantity to which the toxicologist will not often have to confine his analysis in medico-legal practice. Should the quantity of suspected nitrate be very small, it may be placed in a dry Florence flask with a few cuttings of copper and a few drops of strong sulphuric acid poured into the mixture. A slip of paper soaked in a mixture of starch and iodide of potassium may then be suspended by a closely fitting cork in the neck of the flask. Sooner or later, and without the aid of heat, acid fumes will be evolved, and, although the red color may not be apparent, the production of blue iodide of starch in the paper, will indicate their presence and prove that the salt is a *nitrate*. This mode of testing by copper and sulphuric acid is open to objection if any alkaline chloride be mixed with the suspected nitrate. When such a mixture exists (a fact demonstrable by the use of nitrate of silver to a solution of the salt), we may resort to—4. Add a portion of leaf-gold and a few drops of strong and pure hydrochloric acid to the suspected salt in a tube, and warm the mixture by a spirit-lamp. If a nitrate be present the gold is dissolved wholly or in part; and in order to prove that this solution has taken place, a few drops of chloride of tin may be added to the mixture. If any gold is dissolved, the liquid will acquire a pink or dark purple-brown color; otherwise there will be no change of color. The presence of an alkaline chloride does not interfere with this result, but rather aids in the solution of the gold. It must be remembered that a chlorate, bromate, or iodate will dissolve gold under similar circumstances; but the addition of sulphuric acid to these liberates a colored gas or vapor and a peculiar odor. (See 2, p. 211.) The analyst must be careful to use hydrochloric acid free from any trace of nitric acid. It should be tested with gold-leaf previously to adding it to the suspected salt. By the use of either copper or gold, or both, nitric acid or a nitrate, even in minute quantity, may be readily detected.

There are no practical objections which can be urged to the mode of testing for nitric acid above recommended. When the copper and gold-tests yield the results described, the presence of nitric acid or of a nitrate may be considered as conclusively proved.

In Liquids containing Organic Matter.—Nitric acid precipitates and combines with albumen and casein. It may be administered or taken in such liquids as tea, coffee, vinegar, or beer. In this case, besides the acid reaction, there will be a peculiar smell produced by the strong acid, when mixed with substances of an organic nature. Dark-colored liquids are generally made lighter by this acid. The application of the usual tests may be here counteracted: thus, unless the quantity of nitric acid in the liquid is considerable, the orange-red fumes of nitrous acid are not evolved on boiling it with copper cuttings; but the action on leaf-gold will enable a chemist to detect nitric acid in organic liquids, even when the proportion of free acid is very small. Boil a fragment of leaf-gold in pure hydrochloric acid, and add while boiling, a few drops of the suspected organic liquid to the mixture. If nitric acid is present, the gold will be dissolved. This forms a good trial-test.

When the acid liquid is thick and turbid, by reason of its containing blood, mucus, milk, or articles of food of a viscid nature, we may employ the process of dialysis as described for sulphuric acid (see p. 198, *ante*). The vomited matters, or the coats of the stomach and the membrane of the œsophagus cut up and boiled, may be submitted to dialysis. The clear liquid which comes through the dialyzer will be found acid. On neutralizing it with carbonate of potash prismatic crystals of nitre will be obtained on evaporation.

A colored liquid, such as coffee, containing nitric acid, may be at once neutralized with carbonate of potash, filtered and concentrated by evaporation. A few drops of the neutralized liquid may be evaporated on a slide, and the crystals thus obtained microscopically examined and compared with those of nitre. Paper dipped into the concentrated liquid and dried burns with deflagration like touch-paper. The crystals obtained by evaporating the neutralized liquid are generally colored with organic matter, but they fuse into a white mass when gently heated in a platinum capsule. The pure nitre thus obtained may be tested as above described. The organic matter in the crystals does not, however, in any way interfere with the results of the copper and gold tests.

When either the nitric acid, or the nitrate into which it has been converted, is mixed with common salt, the copper test cannot be safely employed. The gold test will in such a case furnish the best evidence. Hydrochloric acid with a small portion of leaf-gold may be added to the dried residue, and the mixture boiled. If nitric acid or a nitrate is present, even in minute proportion, some portion of the gold will be dissolved, a fact demonstrable by the addition of chloride of tin.

Stains on Clothing.—This acid is sometimes maliciously thrown at persons; and we may be required to examine some article of dress suspected to have been stained by it. The spots produced by strong nitric acid on woollen stuffs are either of a yellow, orange-red, or a brown color, according to the time at which they are seen. On black cloth they speedily acquire a light yellowish-brown color, passing after a few days to a dingy olive-green with a red border. After a time they become brown and dry (unlike those produced by strong sulphuric acid), and the texture of the cloth is entirely destroyed. If recent, litmus-paper wetted and pressed upon the spot will indicate acidity. In order to examine them, the stained portions may be cut out and boiled with a small quantity of distilled water. If nitric acid is present, distilled water will acquire an acid reaction; but, in order to prove this, the liquid must be neutralized with potash or its carbonate, and then evaporated to dryness. The dry saline residue, if any, may be examined by the copper and gold tests for nitrate of potash. Should the water acquire no acid reaction, then there is no perceptible quantity of nitric acid present. If the stains are of old date, moistened litmus-paper pressed upon them will give no acid reaction, and no acid liquid will be obtained on boiling the stuff. A simple method of detecting the acid in recent stains consists in boiling a portion of the stained cloth itself with a fragment of gold leaf and hydrochloric acid. If nitric acid is present in the stuff, the gold will be dissolved. An unstained portion of cloth should be at the same time examined.

On these occasions we may be often disappointed in searching for chemical evidence of nitric acid. Not to mention that the acid may be easily removed by washing while the discoloration remains, we must remember that the acid is volatile, easily decomposed, and its nature entirely changed by contact with the organic substance. These facts will explain to us why after a few weeks the chemical evidence of the presence of this acid, is sometimes entirely lost; while in the case of sulphuric acid, the stains may furnish abundant evidence of its presence after many years' exposure.

In all cases of the suspected throwing of nitric acid, the spots on the dress should be examined as soon as possible, or a chemical analysis may be of no avail. The following case occurred at Guy's Hospital: A man had some strong nitric acid maliciously thrown in his face, and the sight of one eye was thereby entirely destroyed. He wore at the time a blue stuff coat, which was not sent to be examined until *five weeks* after the accident, and only a few days before the trial of the prisoner for the offence! The sleeve and body of the coat were found to be covered with numerous spots of a yellowish-brown color. The spots were quite dry; they had evidently been caused by some corrosive acid. The color was discharged, and the fibre of the stuff corroded. Not a trace of nitric acid could be detected in them, although there was no reasonable doubt that it had been used. Its disappearance was probably due partly to its decomposition in the stuff, and partly to its volatility. Had the coat been examined soon after the offence, the nature of the acid would have been easily determined. I have been able to procure certain evidence of the presence of nitric acid in stains on black cloth, a fortnight after the liquid had been spilled. The quantity of acid present was, however, small. Sir R. Christison has obtained evidence of the presence of this acid in the stains on cloth, made seven weeks before (*Op. cit.*, p. 178); and Orfila states that he has found stains on felt, cloth, leather, and even human skin to retain an acid reaction for twelve or fifteen days. He detected nitric acid in the stains, by allowing the material to soak for some hours in a cold weak solution of bicarbonate of soda. The dry saline residue obtained on evaporating the liquid contained a nitrate. (Orfila, *Toxicol.*, vol. 1, p. 187.)

CHAPTER XXVI.

POISONING BY HYDROCHLORIC ACID OR SPIRIT OF SALT—RARELY TAKEN AS A POISON—SYMPTOMS—APPEARANCES AFTER DEATH—FATAL DOSE—CHEMICAL ANALYSIS—DETECTION OF THE ACID IN PURE AND MIXED LIQUIDS—ON ARTICLES OF CLOTHING—IN CASES OF FORGERY.

HYDROCHLORIC ACID.

ALTHOUGH largely employed in the arts, the hydrochloric or muriatic acid is not often taken as a poison. In the Coroners' return for England, during the years 1837–8, out of five hundred and twenty-seven cases of poisoning, there was not one in which this acid was the poison used. Only *three* cases of poisoning by the acid occurred in this metropolis, during a period of sixteen years. Between the years 1863–7, there were eight fatal cases out of 2097 deaths from poison in England and Wales.

There are no doubt many cases in which this acid is taken, but in which it does not prove fatal. These would not be recorded in any registration returns. Dr. Steele informs me that from 1860 to 1874 the admissions into Guy's Hospital of cases of poisoning with this acid were ten, of which one only proved fatal. In one of these cases, a man, æt. 23, swallowed by mistake a wineglassful of strong hydrochloric acid. He suffered from the usual symptoms, but recovered in about six days. (*G. H. Rep.*, 1869, p. 270.)

Symptoms.—From the observations hitherto collected, the symptoms produced by this acid do not differ widely from those caused by the sulphuric and nitric acids. There is the same sensation of burning heat extending from the throat to the region of the stomach, with vomiting of a highly acid liquid of a dark color, mixed with mucus and altered blood. The tongue is swollen and dry; and with much thirst, there is great difficulty of swallowing. The tonsils and throat are inflamed. An escape of acid pungent vapors from the mouth, when the acid has been swallowed, is described by Orfila among the earliest symptoms; after an hour or two this has not been observed. In two cases, neither the vomiting nor pain in the abdomen was urgent, although both terminated fatally. The chief seat of pain was in the throat. In one instance, in which probably an ounce of the acid had been swallowed, the person was able to walk to his home at a distance of three-quarters of a mile. The pulse has been found small, frequent, and irregular; the skin cold and clammy. The intellectual faculties have remained clear until death.

In the case of a Hindoo, Sinivassin, æt. 28, reported by Dr. Collas, the symptoms, about twelve hours after two ounces of the acid had been swallowed, were as follows: the head was drawn backwards, the mouth half open, the lips and face presented no spot or stain, the gums were pale, the teeth not discolored, the tongue was deprived of a strip of its investing membrane about the centre. The skin was cold, the pulse small and frequent, the breathing difficult, the abdomen painful. There was suppression of urine, but no purging. Magnesia with soap and water had been given to him, and were retained on the stomach. It was ascertained that the poison had been taken by mistake for brandy, and that there had been violent vomiting—the vomited matters effervescing on the floor. (*Ann. d'Hygiène*, Janvier, 1858, p. 209.) This case proved fatal. Dr. Procter, of York, communicated to me the particulars of a case in which a woman, æt. 29, swallowed half an ounce of commercial hydrochloric acid. She was seen an hour and a half afterwards. She then complained of intense burning pain in the throat and along the gullet, but there was only slight pain in the stomach, and but very little tenderness of the abdomen. There was incessant vomiting. Magnesia and barley-water were freely given; but in half an hour there was collapse, rendering the use of stimulants necessary. In the evening reaction was established; but the voice could scarcely be heard, and there was great pain in the throat. This was relieved by a few leeches, and the woman recovered in a fortnight. In this instance, the action of the poison appears to have been chiefly spent on the throat and gullet. (*Guy's Hosp. Reports*, 1851, p. 211.) Another case, reported by Dr. Allen, presents a more complete history of the symptoms from a larger dose. A girl, æt. 20, swallowed an ounce of hydrochloric acid on an empty stomach, with the intent to destroy herself. Vomiting had occurred, and alkaline remedies were prescribed before she was seen by Dr. Allen, two hours after the poison had been taken. The countenance was pale and anxious; there was pain with burning heat in the throat and abdomen; the region of the stomach was very tender on pressure, the skin was cold, the pulse 130, small and thready, the tongue pale and whitish, and the throat much inflamed. She vomited freely a fluid of a brownish color, which was quite neutral. Barley-water and carbonate of soda were given. In six hours from the time of taking the poison, she vomited about half a pint of a bloody fluid. Vomiting of blood continued for about twelve hours. On the following day, there was great tenderness in the region of the stomach, with inflammation in the throat and pain in swallowing. In three days there were cramps and twitchings of the limbs, and a sense of coldness in the legs, although these felt quite warm. She then gradually improved; on the fifteenth day the pulse was 80, and she could swallow fluids without difficulty. There was still great tenderness over the stomach. (*Medical Gazette*, 1849, vol. 44, p. 1098.)

In the following case the patient, a woman, æt. 24, did not die for a period of *eight weeks* after taking more than two ounces of this acid (1000 grains). The immediate symptoms were: severe pain with a sense of burning in the tongue, back of the mouth and gullet, as far as the stomach; a feeling of suffocation, escape of white vapors, and vomiting of a liquid which effervesced as it fell on the pavement. In three hours she was brought to the Hôtel Dieu; and it was found that the vomited matters had a brown and bloody appearance. Vomiting continued throughout the night to the extent of four quarts of a reddish liquid, with solid masses of a red-brown color. These vomited matters had no acid reaction; on the next morning, the tongue and throat were covered with a whitish pellicle, and in parts the membrane was removed, as if by the corrosive action of the acid. The inner surface of the cheeks, the roof of the mouth, and under part of the tongue, presented no change. There was a severe burning pain in the throat, extending to the stomach, increased by pressure; but the acid did not appear to have reached the intestines. There was a copious discharge of saliva with shreddy masses of mucus, and any attempt to swallow was followed by spasms in the throat. The voice was feeble and hoarse, breathing quiet, pulse 96, regular and full, skin warm and dry, urine scanty; no evacuation from the bowels. On the second day there was delirium followed by paralysis of the limbs and collapse. During the eight weeks that the patient survived, there were variable symptoms, chiefly referable to the throat, lungs, and stomach. (*Annales d'Hygiène*, 1852, vol. 2, p. 415. Case by Dr. Guérard.)

Appearances after Death.—The throat, larynx, and gullet have been found highly inflamed, the mucous membrane lying in detached masses or actually sloughing away. In one instance the membrane was thickened. The coats of the stomach have been so much corroded that, in many places, there was only the peritoneal coat left; and in attempting to remove the organ in this case, the parietes gave way. The contents have been sometimes of a yellowish, at others of a dark-green color. In a case in which the fundus of the gall-bladder had come in contact with the stomach, it was observed to have a bright-green color, arising from the well-known action of this acid on the bile. On removing the contents of the stomach, the lining membrane has been found blackened, and presenting a charred appearance—the blackening extended through the whole length of the duodenum, and was especially marked on the prominent parts of the numerous valvulæ conniventes (folds of mucous membrane), the intervals being stained of a greenish-yellow color, from the action of the acid on the bile. (Case by Mr. Quekett, *Med. Gaz.*, vol. 25, p. 285.) When death did not take place until after the lapse of several days, the coats of the stomach were of a dark color, highly inflamed, and for the most part in a sloughing state; large dark shreds of membrane were hanging from the sides of the organ, especially about the pylorus. The inflammation had extended also into the duodenum.

M. Tardieu met with the following singular case: A woman applied fuming hydrochloric acid on a pencil to the mouth of a child, fifteen days old, to cure it of some disease. The sucking power of the infant caused a portion of the corrosive liquid to pass down the throat, and thus caused the death of the child. M. Tardieu found the œsophagus deprived of its mucous lining throughout its extent, and covered with false membrane. There were three black patches of corrosion in the stomach. (*Empoisonnement*, 1867, p. 236.)

Perforation of the stomach has not been a common appearance. The mucous membrane of this organ has been found more or less corroded, and sometimes entirely destroyed. In a case referred to by Dr. Galtier (*Toxicologie*, vol. 2, p. 217), which was the subject of a criminal trial in 1856, the stomach was entirely disorganized and softened, and it presented, posteriorly, several perforations of different dimensions, with rounded, thickened, and inflamed margins, adhering to the adjoining viscera by slight albuminous deposits. The pyloric orifice was thickened, as well as the mucous membrane of the small intestines. The large intestines were healthy. The mucous membrane of the throat was thickened, injected, and, on pressure, purulent matter escaped from it. The gullet was thickened throughout its extent, and its mucous membrane was in a state of suppuration. These appearances may be taken as representing the effects produced by the acid when the case is protracted, The patient in this case died eight weeks after taking the acid (p. 216). The quantity taken was unknown. (See also Orfila, *Toxicologie*, vol. 1, p. 216.)

In Dr. Collas's case (*ante*, p. 215), death took place in about twenty-four hours, and the inspection was made thirteen hours afterwards. Although the temperature was high, there was no odor, and no sign of putrefaction. The mouth and throat presented no alteration. The mucous membrane of the tongue was reduced to a grayish pulp, and was easily removed. The membrane of the gullet was rough and disposed in longitudinal folds. The upper and lower portions of the tube were dark-colored, but not carbonized; while the middle portion was pale. The stomach was distended, and presented externally red, green, and black discolorations. It contained about seven ounces of a black turbid liquid. In nearly its whole extent, the surface of the stomach was blackened, and the mucous membrane detached; in the vicinity of the pylorus, it was of a dull wine-red color. The duodenum was healthy, contrasting strongly with the condition of the stomach; this, as well as the other small intestines, contained a yellowish-colored liquid. The cavities of the heart, and the large arteries, contained firm clots of red blood, moulded to the form. The urine was acid, and yielded, by precipitation, a quantity of chloride, indicative, as it was supposed, of the presence of hydrochloric acid = about 0.9 per cent. (*Ann. de Hygiène*, Janvier, 1858, p. 209.)

In Dr. Guérard's protracted case (*ante*, p. 216), in which death took place after eight weeks, the mucous membrane of the gullet was found swollen and softened throughout. At the upper part, the lining membrane was entirely removed; at the lower, it had a slate color. The mucous membrane of the stomach was softened and gelatinized with a brownish discoloration at the greater end; the muscular coat was laid bare in several places. The pylorus (intestinal opening) was hardened, contracted, and of a brown color. The peritoneum was covered with some false membranes. The small intestines were slightly injected. The whole of the parts about the larynx, epiglottis, and trachea were much injected, and of a brown color. A quantity of serum was found in the left pleura, and the lungs were gorged with blood. (*Ann. d'Hygiène*, 1852, vol. 2, p. 423.) For other cases, see Tardieu, *Op. cit.*, p. 241.

Quantity required to destroy Life.—With respect to this question, and *the period* at which the case proves fatal, there is no reason to suppose that the hydrochloric differs from the sulphuric and nitric acids in relation to these points. The cases that have hitherto occurred throw but little light upon the subject. The medical jurist must be content to draw an inference, the fairness of which cannot be disputed, when it is based upon the strong analogy which exists between the effects of this and the other two acids. Dr. Beck states that out of six cases of this kind of poisoning, five proved fatal. (*Med. Jur.*, vol. 2, p. 448.) The facts at present before us are these: In one case, two ounces destroyed life in thirty-three hours; in a second, the same quantity killed a person in eight days; and in a third, a like dose proved fatal *in five hours and a half.* This, I believe, is the most rapidly fatal case on record. The smallest quantity which has been known to destroy life was about half an ounce of the strongest acid. It occurred in King's College Hospital, in May, 1859. A woman, æt. 63, swallowed *half an ounce* of concentrated hydrochloric acid. She was received into the hospital in three-quarters of an hour. The prominent symptoms were burning pain in the throat and stomach, feeble pulse, cold and clammy skin, retching and vomiting of a brown matter streaked with blood, and containing shreds of membrane. There was great exhaustion. The throat became swollen, the patient lost the power of swallowing, and she died in eighteen hours, retaining her senses until the last. The *appearances* in the body were as follows: The mucous membrane of the mouth and throat was white, softened, and destroyed in many places by the corrosive action of the acid. The membrane of the gullet was red and inflamed. The back part of the stomach near the pylorus was black, stripped of its mucous membrane, which was generally softened, and *marked with dark lines.* It was not perforated. (*Lancet*, July 16, 1859, p. 59.) In a case reported by Orfila, the dose was an ounce and a half, and this proved fatal in about eighteen hours. In one case (*ante*, p. 216) a much larger dose did not destroy life until after the lapse of eight weeks. Dr. Otto has published the account of a fatal case in a child in Thorn's *Vierteljahrschrift*, 1865, vol. 1, p. 361. Cases of poisoning by the acid have occurred chiefly among adults; some from accident, and others from suicide.

There have been several recoveries in cases in which an ounce of the acid had been taken. G. S., æt. 23, was admitted into Guy's Hospital in June, 1868. The man had shortly before drunk half a wineglass of strong hydrochloric acid by mistake for brandy. He foamed at the mouth, breathed with difficulty, and was almost asphyxiated. The mouth and throat were clogged with tough viscid mucus. He complained of dryness of the mouth and fauces, and of a severe burning pain in the throat and stomach, but the tongue and mouth were not much affected by the acid. He swallowed with difficulty. The pulse was good, and there was no prostration. The man had vomited several times on his way to the hospital. Albumen and oil were given to him. Thirst and pain in the throat continued for six days. A white layer of membrane came from the tongue. In a week he left the hospital nearly well. (Dr. Stevenson, in *Guy's Hosp. Rep.*, 1869, p. 270. See also the *Lancet* for July 27, 1850, p. 113, *Med. Gaz.*, Dec. 28, 1849, and Beck's *Med. Jour.*, vol. 2, p. 449.)

Hydrochloric acid is not often administered with criminal intention. A trial took place at the Taunton Winter Assizes, 1866 (*Reg.* v. *Somers*), in which a girl of twelve years of age was charged with administering this acid to her mistress in beer, with intent to murder her. Some of the acid had been purchased for domestic use, and the prisoner had been cautioned not to touch it, as it was poisonous. On tasting the beer, prosecutrix perceived an unpleasant taste, and had a burning sensation in her throat. On analysis, the beer was found to contain hydrochloric acid. Life was not endangered, and no grievous bodily harm was done. The prisoner was convicted of a misdemeanor under the new statute, of administering poison with intent to injure, aggrieve, and annoy.

Treatment.—The same as in poisoning by sulphuric and nitric'acids. (See *ante*, p. 194.) It consists in the free use of barley-water, milk, or linseed tea, with carbonate of soda and fluid magnesia.

In general, the mineral acids are taken separately as poisons; but they may be taken in a mixed state; especially as some mixtures of this description are largely used in the arts. Thus, the AQUA REGIA, a mixture of nitric and hydrochloric acids, is used for dissolving gold and platinum; while the AQUA REGINÆ, nitro-sulphuric acid, is employed for dissolving silver and separating it from plated articles. I have not met with any case of poisoning by the nitro-hydrochloric acid; but Orfila gives one case of poisoning by nitro-sulphuric acid. A man, aged 24, swallowed a mixture consisting of one ounce of strong nitric acid and two drachms of strong sulphuric acid. The usual symptoms followed, and he died in eight hours. The appearances, as might have been presumed from the relative quantities of the two acids taken, resembled those of nitric rather than of sulphuric acid. (*Toxicologie Générale*, vol. 1, p. 129.) There is but little doubt that nitro-hydrochloric acid would produce symptoms, and cause appearances, analogous to those described in speaking of hydrochloric acid. The mixed effects of nitric acid might be also perceptible.

CHEMICAL ANALYSIS.

The commercial spirit of salt has a deep lemon-yellow color. It may contain arsenic, antimony, iron, or common salt. It is not always so *concentrated* as to possess the property of fuming in the air; a property which of course depends on its strength, and therefore may be present or absent in any given specimen. A teaspoonful of this acid having a specific gravity of 1.133 was found to weigh 66.4 grains, and a tablespoonful 265.6 grains. The liquid will be found highly acid: it tinges organic substances of a yellowish color, and corrodes them. The specific gravity of the concentrated acid is 1.16, and of the diluted acid of the British Pharmacopœia 1.052. Six fluid drachms contain 36.5 grains of pure hydrochloric acid. 1. The acid, if moderately pure, may be boiled entirely away on pure mercury without being affected by the metal. This experiment serves to distinguish the hydrochloric from the two preceding acids. 2. When boiled with a small quantity of peroxide of manganese, chlorine is evolved, known by its color, odor, and bleaching properties. 3. It does not dissolve leaf-gold until a few drops of nitric acid have been added to it, and the mixture heated. The gold then speedily disappears, and the addition of a small quantity of chloride of tin will show that it is dissolved.

In the *diluted* state, these properties are lost. When the acid is much diluted with water, the property of evolving chlorine with peroxide of manganese, or of dissolving gold, on the addition of nitric acid, is lost. In this case, there is, however, a most satisfactory test for its presence—the *nitrate of silver*. This test gives, with the acid, a dense white clotted precipitate of chloride of silver. The precipitate thus formed acquires speedily a dark color by exposure to light; and it is known from all other white salts of silver, by the following properties: 1. It is insoluble in nitric acid. 2. It is soluble in caustic ammonia. 3. When dried, and heated on platinum, glass, or mica, it melts like a resin, forming a yellowish-colored sectile mass. Unless these properties are possessed by the precipitate, it is impossible to refer the action of the test to the presence of hydrochloric acid.

In Liquids containing Organic Matter.—If hydrochloric acid is present in a noxious proportion, they will have an intensely acid reaction. Many liquid articles of food—such as wine, beer, cider, or vinegar—have an acid reaction, and frequently contain an alkaline chloride. A mixture of lemon-juice and common salt, which might be present in the contents of the stomach, would give the chemical reactions of diluted hydrochloric acid with the tests above described, and the natural mucous secretions of the stomach contain hydrochloric acid, with alkaline chloride. Hydrochloric acid may be present, irrespective of poisoning, in the decoction of the coats of the stomach and œsophagus, or in the matters which have been vomited. If the acid is found only in minute quantity, no inference of poisoning can be drawn, unless there are distinct marks of its chemical action upon the throat and stomach. The presence of local chemical changes from the throat to the stomach, would show whether the acid had been taken as a poison, or not. When no more than slight traces of acidity are found with a quantity of alkaline chloride, no reliance can be placed on the chemical results. In two trials for murder, which occurred in France (Orfila, *Toxicologie*, p. 216), and in one which occurred in Belgium, in which the acid was given with a view to procure abortion (*Galtier*, vol. 1, p. 217), the evidence respecting the presence of poison in the bodies failed on this ground, although the appearances left no doubt that a corrosive liquid had been taken. The analysts too strongly relied on the nitrate of silver as a test, without having had due regard to the presence of alkaline chlorides in the fluids of the stomach. On this account the chemical evidence was rejected as unsatisfactory, but, as in the abortion cases the women did not die until after the lapse of two months, it would have been something unusual had any of this poison remained in the body. (The reader will find a report of these cases in Flandin's *Traité des Poisons*, vol. 2, pp. 482, 491.)

Full allowance must therefore be made for the natural presence of chlorides in acid liquids on these occasions. They may always be detected by evaporating the liquid and incinerating the residue.

If the acid liquid contains much organic matter, such as milk, mucus, blood, or other substances of a viscid nature, it may be submitted to dialysis (*ante*, p. 198), like sulphuric acid, and having obtained thereby a clear acid liquid, the silver and other tests for the diluted hydrochloric acid may be applied to it. The amount of precipitate obtained would furnish an indication of the proportion of free acid present, and the evaporation of a similar portion of it to dryness, would show whether any part of this precipitate was really due to the presence of alkaline chloride. M. Bouis has suggested that the presence of free hydrochloric acid may be detected by boiling in the liquid a portion of leaf-gold and nitrate, or chlorate of potash. If the free acid is present the gold will be dissolved; if there is only a chloride, the metal will remain undissolved. (*Ann. d'Hyg.*, 1874, vol. 1, p. 458.)

When the hydrochloric acid contained in the organic liquid is in moderately large quantity, I have found that it may be obtained by distilling the liquid to dryness in a sand-bath. The distillate in the receiver readily shows the presence of hydrochloric acid. This process has the advantage of separating the free acid from any chloride associated with it. It is not applicable to those cases in which the acid liquid contains much organic matter, but to these the process of dialysis may be readily applied. The acid obtained by either of these methods is, of course, always in a diluted state. The concentrated acid is only met with as a residue in any vessel out of which it may have been taken.

On Articles of Clothing.—Chemical evidence may be obtained from this source when other sources fail. In a case which occurred to Mr. Quekett the acid was not found in the stomach; but the nature of the poison was accurately determined by examining a portion of the deceased's waistcoat, on which some of the acid had become accidentally spilled.

By digesting the stained stuff in warm distilled water, a highly acid liquid may be found on filtration, which, if hydrochloric acid be present, will yield, with nitrate of silver, a white precipitate, possessing all the properties of chloride of silver. Another method of testing may also be applied when the stain from the strong acid is recent. Leaf-gold should be boiled in strong nitric acid and a portion of the stained cloth added. If hydrochloric acid is present, the gold will be dissolved, otherwise not.

The acid by exposure of the cloth soon passes off, as it is more volatile than the other acids. The cloth then becomes dry, and on pressing wet litmus-paper on the stain, there will be no acid reaction. The spots produced on black cloth by the strong acid are at first of a dark crimson red, but in ten or twelve days they change to a red-brown. Hence it will be perceived that this acid differs from the others in the effect produced on black cloth. Sulphuric and nitric acids produce brown and not red stains, the stain from the former acquiring a red fringe only after some days. An unstained portion of the cloth should always be examined by way of comparison. The red stain produced by the acid on black cloth is removed by boiling water, the cloth becoming black, but again on drying acquiring a red-brown color. The diluted hydrochloric, like the diluted sulphuric and nitric acids, produces at once red stains on black cloth.

If this acid has been used in the erasure of writing ink for the purposes of *forgery*, its presence in the paper may be detected by a similar process. Supposing that there should be no free acid in the paper, the addition of ferrocyanide of potassium (by producing Prussian blue) will show that a soluble salt of iron (sesquichloride) has been diffused through the substance of the paper. A man of the name of Hart was tried at the Central Criminal Court, Dec. 1836, on a charge of forgery, under the following circumstances. The prisoner received a blank acceptance for £200, and afterwards erased the figure 2 by an acid and substituted the figure 5. The witness who gave chemical evidence on this occasion, deposed that some acid had been used to effect the erasure, but he could not ascertain its nature. He suspected that it must have been either the hydrochloric or oxalic acid, probably the former. Counsel ingeniously objected to the evidence, that chloride of lime was used in the manufacture of the paper and might account for the results obtained by the silver test; but in answer to this, it was properly stated, that the chloride of lime was entirely removed by subsequent washing. If any acid liquid were obtained from a stain on paper under these circumstances, the hydrochloric would be easily known from the oxalic acid by the fact that the chloride of silver is not soluble in nitric acid, while the oxalate of silver is soluble in it.

CHAPTER XXVII.

POISONING WITH OXALIC ACID—SYMPTOMS AND EFFECTS—APPEARANCES AFTER DEATH—ITS LOCAL ACTION ON THE STOMACH—PERFORATION OF THE COATS—FATAL DOSE—RECOVERY FROM LARGE DOSES—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT—CHEMICAL ANALYSIS—TESTS FOR OXALIC ACID IN PURE AND MIXED LIQUIDS—OXALIC ACID IN ORGANIC SUBSTANCES—POISONING BY THE RHUBARB OR PIE PLANT—QUANTITATIVE ANALYSIS.

OXALIC ACID.

OXALIC ACID is one of the most powerful of the common poisons; but its use as a poison is almost entirely confined to this country. Cases of poisoning by it are generally the result of suicide or accident. In the Coroners' return for 1837–8 there were nineteen cases of poisoning by this substance, out of which number fourteen were the result of suicide. It is singular, also, that the greater number of these cases occurred in the County of Middlesex. In the later return, 1863–7, 66 fatal cases were recorded. In Guy's Hospital, from 1860 to 1874, there were fourteen cases of poisoning with this acid, not one of which proved fatal. Accidental poisoning by oxalic acid has frequently arisen from its strong resemblance to Epsom salts. It is not often that we hear of it being used as a poison for the purposes of murder. Its intensely acid taste, which could not be easily concealed by admixture with any common article of food, would infallibly lead to detection long before a fatal quantity had been swallowed. I have known several trials to take place for attempted poisoning by oxalic acid—in two the vehicle selected for its administration was coffee or tea, and in one the poison was powdered and mixed up with brown sugar to conceal the taste. (*Reg.* v. *Dickman*, Central Criminal Court, February, 1845.) In another, buttermilk is supposed to have been the vehicle of the poison.

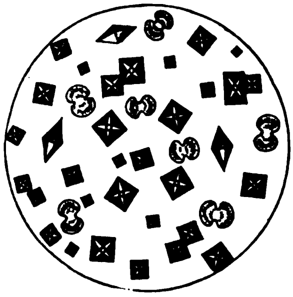
Symptoms.—In some cases of poisoning by this substance death has taken place so rapidly that the person has not been seen alive by a medical practitioner. If the acid is taken in a large dose, *i. e.*, from half an ounce to an ounce of the crystals dissolved in water, a hot, burning, sour taste is experienced in the act of swallowing, extending downwards to the stomach, and vomiting occurs either immediately or within a few minutes. There is also a sense of constriction in the throat, almost amounting to choking or suffocation. Should the poison be diluted there is merely a sensation of strong acidity, and vomiting occurs only after a quarter of an hour or twenty minutes. In some instances there has been little or no vomiting; while in others this symptom has been incessant until death. Thus, in a case in which an ounce of the acid was swallowed, vomiting, with pain in the stomach, continued until the fifth day, when the man died suddenly (*Lancet*, Nov. 24, 1860, p. 509); but in another, in which the poison was much diluted, vomiting did not occur for seven hours. (Christison, *Op. cit.*, p. 221.) The vomited matters are highly acid, and have a dark brown or almost black appearance; they consist chiefly of mucus and altered blood. In a case which occurred to the late Dr. Geoghegan they were colorless (*Med. Gaz.*, vol. 37, p. 792); and in another, fluid blood of a bright arterial color was vomited after some hours. (*Provincial Journal*, June 25, 1851, p. 344.) There is at the same time a burning pain in the stomach, with tenderness of the abdomen, followed by close clammy perspiration and convulsions. In another case that occurred in Guy's Hospital, in May, 1842, in which about two ounces of the poison had been swallowed, there was no pain. Urgent vomiting and collapse were the chief symptoms. There is in general an entire prostration of strength, so that if the person be in the erect position he falls; there is likewise unconsciousness of surrounding objects, and a kind of stupor, from which, however, the patient may be without difficulty roused. Owing to the severity of the pain the legs are sometimes drawn up towards the abdomen, or the patient rolls on the floor or bed. The pulse is small, irregular, and scarcely perceptible; there is a sensation of numbness in the extremities, and the breathing, shortly before death, is spasmodic. The inspirations are deep, and a long interval elapses between them. In one case the patient was found insensible, and the jaws spasmodically closed. Such are the effects commonly observed in a rapidly fatal case.

The symptoms have been described as occurring *immediately* in this form of poisoning. This may give rise to an important question. The effect on the tongue and throat, as well as on the stomach, cannot be concealed, even if the vomiting and pain should not occur for some time; but in the cases of children there may be a difficulty in drawing a conclusion. In *Reg.* v. *Cochrane* (Liverpool Summer Assizes, 1857), a woman was indicted for administering to two of her children oxalic acid in half a pint of buttermilk. They were found dead in two hours, under circumstances of great suspicion; the body of one was cold. The room was full of smoke, owing to its having apparently been set on fire. The bodies were separately examined by different surgeons; one came to the conclusion that the child whose body he had examined had died from suffocation, the other that the second child had died from corrosive poison. The mucous coat of the stomach in the child was inflamed and softened, so that it readily gave way. There were two brownish-colored patches, and the submucous coat was exposed. The stomach contained a fluid which, when examined by Dr. Edwards, yielded oxalic acid to the amount of about forty-two grains. In the stomach of the other child, supposed to have been suffocated, twenty grains were found. There is no evidence that the children had vomited, and it is not improbable that death took place rapidly. The woman was acquitted. There was a difficulty in reference to the administration of the poison in this case, which has not before presented itself. A large quantity of the acid must have been dissolved in half a pint of buttermilk, to have destroyed these children, and have left so large a residue in the stomach. The children (æt. six and four respectively) were supposed to have swallowed this intensely acid liquid without difficulty or complaint, and without any of the usual symptoms being produced! The following case is exceptional, from the fact that the symptoms throughout were chiefly referable to the brain. A man took what was supposed to be a black draught, but it contained oxalic acid instead of Epsom salts. Two hours afterwards he was found in a state of complete coma, but the symptoms set in in a quarter of an hour after he had taken the draught. The man died in five hours, without recovering his consciousness. The only marked appearance, on inspection, was intense congestion of the brain. (*Lancet*, 1872, vol. 2, p. 41.)

The urine which is passed by a person laboring under the effects of this poison, will be found after a few hours to deposit a sediment in which the well-known octahedral crystals of oxalate of lime may be seen by the use of the microscope. (Fig. 8.) M. Tardieu recommends this as an aid to diagnosis. (*Empoisonnement*, 1867, p. 252.)

Should the patient survive the first effects of the poison, the following symptoms appear: There is soreness of the mouth, constriction and burning pain in the throat, with painful swallowing; tenderness in the abdomen, with irritability of the stomach, frequent vomiting, accompanied by purging. The tongue is swollen, and there is great thirst. A patient may slowly recover from these symptoms, but he may die many days afterwards from starvation as a result of the destructive action of the poison on the œsophagus and stomach. In a protracted case of this

FIG. 8.



Deposit of oxalate of lime from the urine.

kind, which occurred to Mr. Fraser, the following symptoms were observed: A gentleman by mistake swallowed half an ounce of oxalic acid dissolved. He felt immediate irritation in the throat and stomach. He swallowed some water, which was followed by vomiting. When seen soon afterwards, he complained of excruciating pain, and had violent spasms. The tongue was swollen and covered with a thick white coat as if it had beeen scalded. There was difficult breathing, general numbness, with clammy moisture on the skin; pulse scarcely perceptible; limbs cold and nails livid. The vomited matters were tinged with blood, and a large quantity of blood was brought up. There were spasms, with painful numbness and great loss of strength. The symptoms of irritation abated, and he appeared to have recovered from the direct effects. On the fourteenth day after taking the poison, he died evidently from starvation, as a result of the local action of the poison. (*Ed. Med. Jour.*, vol. 14, p. 607.)

In a case reported by Mr. Edwards, the patient, a woman, lost her voice for eight days. In a former edition of this work, I treated it as doubtful whether the loss of voice could have depended on the action of the poison. A case has, however, since occurred to Mr. T. W. Bradley, from which it may be inferred that a loss of voice may result from the direct effect of oxalic acid on the nervous system. A man swallowed a quarter of an ounce of the acid, and suffered from the usual symptoms in a severe form. In about nine hours, his voice, although naturally deep, had become low and feeble. This weakness of voice remained for more than a month, and its natural strength had not returned even after the lapse of nine weeks. During the first month there was numbness, with tingling of the legs. (*Med. Times*, Sept. 14, 1850, p. 292.) The occurrence of this sensation of numbness, and its persistence for so long a period after recovery from the symptoms of irritation, clearly point to a remote effect on the spinal nervous system. Spasmodic twitchings of the muscles of the face and extremities have also been observed in some instances. (See *Lancet*, March 22, 1851, p. 329.)

From Sir R. Christison's experiments, it would appear that this acid is still a poison, even when so diluted as to lose all its irritant and corrosive properties. It thus differs from the mineral acids. The effects vary according to the quantity. In a large dose, but much diluted, the poison, he considers, destroys life by producing paralysis of the heart. When the dose is diminished, the spinal marrow is affected, and tetanus is one of the symptoms; when still less, but enough to prove fatal, the poison acts like a narcotic, and the animal dies as if destroyed by opium. (*On Poisons*, p. 219.) Dr. Pelikan, Professor of Medical Jurisprudence at St. Petersburg, informs me that these variable modes of operation on animals, as a result of dilution, are not in accordance with his observations. Even when taken in the solid state, oxalic acid operates with rapidity. In July, 1874, a man was admitted into the Westminster Hospital, who had shortly before taken a quantity of solid oxalic acid. It seems he had eaten it on his way to the hospital. In spite of treatment, he rapidly became pulseless, sank into a state of collapse, and died in less than an hour after admission. (*Pharm. Jour.*, 1874, p. 96.)

Appearances after Death.—The lining membrane of the mouth, tongue, throat, and gullet is commonly white and softened, but often coated with a portion of the dark-brown mucous matter discharged from the stomach. Sometimes the membrane has presented a bleached appearance. The *stomach* contains a dark-brown mucous liquid, often acid, and having almost a gelatinous consistency. On removing the contents, the mucous membrane will be seen pale and softened, without always presenting marks of inflammation or abrasion, if death has taken place rapidly. This membrane is pale, soft, and brittle, easily removed, and presents that appearance which we might suppose it would assume, if it had been for some time boiled in water. The small vessels are seen ramifying over the surface, filled with dark-colored blood, apparently solidified within them. The lining membrane of the gullet presents much the same characters. It is pale, and appears as if it had been boiled in water, or digested in alcohol; it has been found in longitudinal folds, interrupted by patches where the membrane had been removed. In a case which was fatal in eight hours, the tongue was dotted with white specks; the gullet was not inflamed, but the stomach was much destroyed, and had a gangrenous appearance. Portions of the mucous membrane were detached, exposing the muscular coat. With respect to the intestines, the upper portion of the canal may be found inflamed; but unless the case be protracted, the appearances in the bowels are not strongly marked. In a case of poisoning by this acid, however, which is recorded by Dr. Hildebrand, the mucous or lining membrane of the stomach and duodenum was very strongly reddened, although the patient, a girl of eighteen, died in three-quarters of an hour after taking one ounce of the acid, by mistake for Epsom salts. (Casper's *Vierteljahrschrift*, 1853, 3 B., 2 H., p. 256.) In another case, in which two ounces of the acid had been taken, and death was rapid, the coats of the stomach presented almost the blackened appearance produced by sulphuric acid, owing to the color of the altered blood spread over them. In protracted cases the œsophagus, stomach, and intestines have been found more or less inflamed. In a case in which an ounce was swallowed and death occurred on the fifth day, the stomach was slightly congested and contained a bloody fluid, but the mucous membrane was entire.

In Mr. Fraser's case (*supra*, p. 226), in which death took place on the *fourteenth day* from starvation, the following appearances were met with: The body was inspected fifteen hours after death. There was great emaciation. The stomach contained a small quantity of dark-colored fluid. Its inner surface, as well as that of the intestines, showed marks of inflammation. The mucous coat was entirely destroyed, as well as that of the œsophagus, so that the muscular coat was laid bare. In some parts it seemed entire, but on examination it was found to be soft and easily detached by the finger or sponge. The muscular coat of the stomach and œsophagus was much thickened, highly injected, and had a dark gangrenous appearance. There was no perforation, but the cardiac was more inflamed than the pyloric end. The small intestines presented a similar appearance, but in a much slighter degree. (*Ed. Med. Jour.*, vol. 14, p. 607.)

I am indebted to Mr. Welch for the particulars of a case of poisoning by oxalic acid which occurred in April, 1853. A woman, æt. 28, swallowed *three drachms* of the crystallized acid. She was found quite dead in *one hour* afterwards. On examining the body, both lungs were observed to be extensively congested, and the heart and large vessels were full of dark-colored blood. The stomach contained about three-quarters of a pint of dark-brown fluid, and its lining membrane was generally reddened. The other organs, excepting the brain, were healthy, and this presented appearances indicative of long-standing disease. There was serous effusion, with great congestion of the vessels. This case is remarkable from the smallness of the dose, the rapidity of death, and the well-marked redness of the mucous membrane of the stomach. The diseased state of the brain may have tended to accelerate death from the poison in the stomach. In one instance the larynx was filled with frothy mucus, and the left side of the heart and the lungs were gorged with dark-colored fluid blood. In another, the appearances of the sanguineous apoplexy were found in the brain. The patient fell dead after retching violently. Apoplexy was supposed to be the cause of death. On an inspection of the body, it was found that a large clot of blood was effused on the brain, and this appeared to account for death satisfactorily. But when the stomach was examined, oxalic acid was detected in it. This poison had been taken with suicidal intention, and had produced its usual effects. The violent vomiting which it caused had led to death by apoplexy from effusion of blood on the brain. (*Lancet*, 1863, vol. 1, p. 47.) Without a proper chemical investigation, it is obvious that the real cause of death would have been in this instance overlooked. In a few cases there have been scarcely any morbid appearances produced by the poison.

It is worthy of remark that the glairy dark-colored contents of the stomach do not always indicate strong acidity until after they have been boiled in water.

Oxalic acid does not appear to have a strong corrosive action on the stomach, like that possessed by the mineral acids. It is, therefore, rare to hear of the coats of this organ being perforated by it. In experiments on animals, and in some observations on the human subject, I have found nothing to bear out the view that perforation is a common effect of the action of this poison. The acid undoubtedly renders the mucous coat soft and brittle, and it dissolves by long contact animal matter, which on analysis is found to be of a gelatinous nature. Its solvent powers on the animal membranes are not, however, very strong, as the following experiment will show. A portion of the jejunum of a young infant cut open, was suspended in a cold saturated solution of oxalic acid for six weeks. At the end of this time, the coats, which were white and opaque, were well preserved, and as firm as when they were first immersed, requiring some little force with a glass rod to break them down.

Sir R. Christison refers to only one instance in which, after death from oxalic acid, the stomach was found perforated. Dr. Letheby has reported the following case. An unmarried woman, æt. 22, of previously good health, swallowed one evening, a dose of oxalic acid (quantity not known), and the next morning she was discovered dead in her room. On inspection, the stomach was found much corroded and softened. The mucous membrane was much blanched, except in two or three places, where there were small black spots, as if blood had been effused and acted upon by the poison; and here and there a blood-vessel might be seen ramifying, with its contents similarly blackened. The coats of the stomach were so softened, that it could scarcely be handled without lacerating it. At the cardiac end the coats were of a pulpy or gelatinous consistency, and presented numerous perforations. The contents amounted to six ounces, and were of a dark color like porter, with but little solid matter. The liquid was strongly acid; and on being tested was found to contain about three drachms (180 grains) of oxalic acid. The softening effect here was probably due to long contact of a large quantity of the acid after death. (*Med. Gaz.*, vol. 35, p. 49.) In a case which occurred to Dr. Wood in May, 1851 (*Ed. Month. Jour.*, March, 1852, vol. 14, p. 227), the stomach was found perforated. The deceased, a nurserymaid, æt. 27, was found dead on her right side, the knees drawn up to the abdomen, and the right arm was slightly extended. Dr. Wood was informed, that shortly before he was sent for she had vomited, and was unable to speak. (Some acid crystals were found in a saucer in the room.) It seems that while vomiting she fell on the floor senseless. On inspecting the body, thirty-five hours after death, there was a frothy liquid around the mouth, with minute acicular crystals. The tongue, pharynx, and gullet had a blanched appearance; the gullet a bluish leaden-gray color, and the membrane was easily removed by a scalpel. The stomach presented a large irregular aperture at its upper and forepart, nearer the cardia than the pylorus. From this a dark gelatinous-looking matter, resembling coffee-grounds, escaped in abundance. The aperture, before handling, was of a size to admit the point of the finger; but it was enlarged by removal. It eventually had the appearance of two large apertures separated by a narrow band. The internal surface of the stomach was occupied by the same grumous-looking fluid, and the mucous membrane had an eroded appearance. The small intestines presented changes of a similar character. The larynx was filled with a frothy mucus. The heart and lungs were healthy. The left cavities of the heart as well as both lungs were gorged with blood. Oxalic acid was found in the vomited matters, and in the contents of the stomach. The acid had produced yellow spots on the cloth boots of the deceased.

The late Dr. Geoghegan has given the following account of the appearances met with in the stomachs of three persons poisoned by oxalic acid. The first was taken from the body of a young man, who must have died in about twenty minutes after swallowing the poison. The inspection was made the following day. The stomach contained eight ounces of a dark-brown and viscid matter, resembling coffee-grounds, evidently largely impregnated with altered blood, and possessing an acid reaction. The mucous membrane at the larger end (cardia) was of a deep blackish-brown color, of variable intensity. The discolored condition of the membrane extended in narrow streaks into the body of the organ, where the lining membrane was otherwise of a uniform light purple-red color. Near the pylorus the membrane was translucent, and exhibited the dark ramiform vascularity of the submucous coat. The mucous membrane of the cardiac portion was soft and thin, detachable only as a pulp, and in parts eroded. In the body of the stomach the lining membrane was somewhat thickened, but less soft, removable in flakes of one-third of an inch; at the intestinal end not thickened, and yielding strips of one-fourth of an inch. In reference to these three cases, Dr. Geoghegan observes: Although in one of them, the contents, including no inconsiderable amount of acid, remained in contact with the coats of the organ, no perforation was observable, the solvent energies being diffused over a large surface. The dose was not ascertained in any of the cases. Oxalic acid and gelatin were discovered readily in the contents in all. The quantity of poison in the contents was, in the first case, about three to four grains; there was a larger quantity in the two latter, especially in No. 3.

A comparison of these cases with those already on record gives as the ordinary appearances in the stomachs of persons who have succumbed to the influence of oxalic acid—1. Contents, of the color of coffee-grounds, consisting of altered blood and mucus, and separating into a supernatant fluid and insoluble deposit. 2. Softening of mucous membrane, with various shades of brown coloration, erosion, or gelatinization. 3. Brownish-black ramiform vascularity of the submucous tissue, owing to the imbibition of the acid contents. It is important to note in similar cases the coexistence of this latter condition with the state of the contents just described, as the ramiform vascularity, or diffuse brown discoloration, presents itself in many instances as a result of the action of lactic acid contained in the gastric juice. It appears evident that the fatal result in cases of poisoning by oxalic acid cannot be referred to the corrosion of the stomach as its chief cause, but rather to the contemporaneous and energetic action which it exerts by absorption in arresting the circulation. (*Med. Gaz.*, vol. 38, p. 792.) The œsophagus, stomach, and bowels, have been chiefly examined in these inspections. Dr. N. Chevers describes the case of a man found dead at Mooltan, in September, 1853, with his right hand clasped over his stomach, showing a very sudden death. There were no marks of external violence, but there were appearances of convulsions about the fingers and mouth. The stomach was found in a state of contraction from spasm. The mucous surface at the lower part was greatly and recently inflamed, and a minute quantity of oxalic acid was detected in the contents. The cerebrum and cerebellum were much congested. There was a slight effusion of serum beneath the pia mater (inner membrane of the brain), but no actual effusion of blood had occurred. (*Medical Jurisprudence for India*, p. 165.) The medical officer was inclined to refer death to congestion of the brain; but it was clearly due to oxalic acid—the congestion being probably one of the effects produced by the poison. In a case in which I was consulted in December, 1854, a large dose of oxalic acid proved fatal in two hours. With the usual appearances in the stomach, the head and chest presented nothing unnatural. The heart contained dark fluid blood in all its cavities.

Fatal Dose.—On a trial for murder by this poison, a question arose respecting the quantity required to destroy life. One witness deposed, that he thought ten grains of the acid was sufficient; another said that it was not sufficient. The prisoner was acquitted. A question of this kind can be solved only by a reference to recorded facts; but unfortunately, in most cases, it has been impossible to determine exactly the quantity of poison taken. Oxalic acid, it is to be observed, presents some singular anomalies in its effects. In one case a man swallowed, as nearly as could be ascertained, three drachms of the crystals: there was immediate vomiting, but no other urgent symptoms, and he recovered in a few hours. In a second instance, a woman took nearly half an ounce of the acid—the usual symptoms appeared—she recovered in six days, and was able to leave the hospital. Mr. Semple met with a case, where a girl swallowed about two drachms of the poison dissolved in water. Vomiting occurred immediately. In about twelve hours the more urgent symptoms had disappeared; but there was still tenderness of the abdomen with irritability of the stomach. In the course of a few days the patient was quite well. In February, 1842, a case occurred at King's College Hospital, where a girl had swallowed two drachms of the acid, dissolved in beer. The only symptom from which she suffered on admission, was pain. She entirely recovered the next day. Dr. Babington, of Coleraine, has reported a case (*Med. Gaz.*, vol. 27, p. 870), in which a girl swallowed by mistake two scruples (*forty grains*) of the poison. Severe symptoms followed, chiefly marked by great irritation of the stomach. It was a week before this girl had recovered, and a much longer time elapsed before she was able to resume her duties. In these cases, it is to be observed, proper medical treatment was resorted to; and the effects of the poison may be therefore supposed to have been in a great degree counteracted. But this explanation is hardly sufficient to meet such cases as the following. A girl, æt. 15, swallowed two pennyworth (half an ounce) of oxalic acid, and she was not admitted into St. Thomas's Hospital until half an hour afterwards; a period had therefore elapsed, within which death has frequently taken place. When admitted she complained of great heat, and a sense of burning about the throat and fauces, with a feeling of sickness at the pit of the stomach; she vomited a large quantity of bloody frothy mucus. The stomach-pump was used, and some prepared chalk in water was injected. After this she appeared sinking; signs of collapse came on; the blood left the surface; the extremities were cold, and the pulse was hardly perceptible. Stimulants were given, and artificial warmth applied. The next day there was great soreness of the mouth and tongue, and the latter was swollen, red, and tender; skin hot; tenderness on firm pressure of the stomach. In a few days she perfectly recovered. (*Med. Gaz.*, vol. 1, p. 737.) In the summer of 1846 I saw a similar case, in which a like quantity had been taken by a patient in Guy's Hospital; and here the extremities were cold; but there was little pain on pressure of the abdomen some hours after the poison had been taken. The woman recovered.

It is not improbable that idiosyncrasy may account for these anomalies: *i. e.*, that certain constitutions are with difficulty affected by this poison. Two cases have occurred at Guy's Hospital, in each of which half an ounce of oxalic acid had been swallowed. Active treatment was adopted, and both patients recovered.

When the dose is upwards of half an ounce, death is commonly the result; but one of my pupils informed me of a case in which a man recovered, after having taken *an ounce* of crystallized oxalic acid; and Dr. Brush, of Dublin, has communicated to the *Lancet*, a case in which perfect recovery took place after a similar dose of the poison had been taken. The acid was in this instance taken by mistake for Epsom salts. One ounce was put into a tumbler, and boiling water was poured on it at night. About half-past four in the morning, the patient, a man aged sixty, stirred up the liquid and swallowed the whole. Contrary to what has been hitherto observed, there was no *immediate* vomiting; the man, having discovered his mistake, tried to excite it, and only partially succeeded after the lapse of ten minutes. Warm water was freely given to him, and he ejected from his stomach dark clotted blood mixed with mucus. The usual antidotal treatment was then resorted to, and the stomach-pump used. In two hours symptoms of collapse appeared. In about six hours the skin had regained its warmth; but there was no pain in the stomach or any part of the abdomen. The secondary symptoms were a burning sensation in the mouth and throat, great difficulty in swallowing, thirst, acid eructations, and drowsiness; and these symptoms continued for two or three days. Vomiting and irritability of the stomach remained until the sixth day, but from this time the recovery was rapid; and in about eighteen days all unfavorable symptoms had disappeared. (*Lancet*, July 11, 1846, p. 39.) In the same journal is reported another case of recovery after an *ounce* of the acid had been swallowed. The man, it is stated, was not seen until fourteen hours after he had taken the poison; and he had, in the meantime, travelled a distance of ten miles to Dublin. He had immediately taken warm water. On his arrival in Dublin, magnesia and rhubarb were given to him. He complained of a burning sensation in the throat and gullet; his tongue was coated, and his pulse was small, quick, and wiry. There was anxiety of countenance, with complete prostration of strength. The palate was blistered, and the throat was highly inflamed; there was tenderness of the stomach, with vomiting of a dark substance mixed with blood. The man ultimately recovered, but for a long time afterwards he complained of a sense of constriction in the œsophagus. (*Lancet*, Sept. 13, 1845, p. 293.) The reporter of this case states, that the quantity of the poison actually taken, exceeded an ounce. (See also cases by Mr. Allison, *Lancet*, Nov. 2, 1850, p. 502, and by Dr. Barham, *Prov. Med. Journ.*, Oct. 6, 1847, p. 544.) Dr. Ellis met with a case of recovery in a woman, æt. 50. She took an ounce of the acid in beer. In half an hour she was found rolling about and complaining of a burning pain in the stomach. Chalk and water were freely given to her, and she recovered. (*Lancet*, Sept. 3, 1865, p. 265.)

According to the experiments of Mitscherlich, two drachms of the acid killed a rabbit in a quarter of an hour, and half a drachm killed another in half an hour. Fifteen grains produced general disturbance of the functions, but did not prove fatal. It is strange that this experimentalist should assert that oxalic acid does not produce inflammation of the intestinal canal. There are several cases recorded in this chapter which prove that this is a mistake—another instance of the fallacies of "animal" experience. The smallest fatal dose of this poison yet recorded, is *one drachm* or sixty grains. This fell under the observation of the late Dr. Barker, of Bedford. (*Lancet*, Dec. 1, 1855.) He ascertained, on inquiry, that a boy, æt. 16, bought half an ounce of oxalic acid; he took about a quarter of it, eating it as a dry solid, and threw away the remainder. He was found in about an hour insensible, pulseless, and with the jaws spasmodically closed. He had vomited some bloody matter; his tongue and lips were unusually pale, but there was no excoriation. He died within nine hours after taking the poison.

It may be proper to state, that this poison is retailed to the public at the rate of from a quarter to half an ounce for one penny or twopence, and one ounce for twopence or fourpence.

Period at which Death takes place.—Similar quantities of this poison do not destroy life within the same period of time. In two cases, in which about two ounces of the acid were respectively taken, one man died in twenty minutes, the other in three-quarters of an hour. Sir R. Christison mentions an instance in which an ounce killed a girl in thirty minutes; and another in which the same quantity destroyed life in *ten minutes*. In Mr. Welch's case (p. 228) three drachms destroyed life in an hour.

The late Dr. Ogilvy, of Coventry, has reported a case of poisoning by oxalic acid, in which it is probable that death took place within *three minutes* after the poison had been swallowed. The sister of the deceased had been absent from the room about this period, and on her return, found her dying. The quantity of poison taken could not be determined. The only other remarkable circumstance in the case was, that the coats of the stomach were so softened, that on an attempt being made to remove the organ, they were lacerated by the weight of the contents. The intestines and left lobe of the liver were also found softened, as if by transudation. This is the most rapidly fatal case on record. (*Lancet*, Aug. 23, 1845, p. 205; and *Med. Gaz.*, vol. 36, p. 831.) The softening of the stomach was, no doubt, a post-mortem effect of the acid. Dr. Iliff communicated to me the particulars of a case in which the wife of a druggist, who had taken a dose of oxalic acid, was found dead by the side of the counter within a few minutes after she had been seen living. The stomach contained a black viscid acid liquid. The mucous membrane was not destroyed, and there were no particular signs of inflammation. The veins were gorged with blood, which gave a peculiar appearance. The tongue was white, but neither the throat, gullet, nor alimentary canal presented any marks of inflammation. The vessels of the brain were turgid, and the pupils were dilated. In these rapidly fatal cases, the poison is supposed to operate by causing paralysis of the heart. When the dose of oxalic acid is half an ounce or upwards, death commonly takes place within an hour. There are, it must be admitted, numerous exceptions to this rapidity of action. Sir R. Christison reports two cases, which did not prove fatal for thirteen hours. In the case which occurred to Mr. Fraser, in which only half an ounce was taken, the person died from the secondary effects, in a state of perfect exhaustion, *fourteen days* after taking the poison. (See p. 227, *ante.*)

Treatment.—It is recommended that water should be sparingly given, as it is apt to lead to the more complete solution, diffusion, and absorption of the poison. But in some instances water has been productive of great benefit, and has aided the efforts of the stomach to expel the poison by vomiting. (See the case by Dr. Brush, *ante*, p. 232.)

The best remedies are: the saccharated solution of lime, or precipitated chalk made into a cream with water or milk, and administered in small quantities at short intervals. If much fluid has been swallowed, the stomach-pump may be resorted to, and the stomach well washed out with lime-water. The poison in many instances acts with such rapidity, as to render the application of these remedies a hopeless measure. The use of the alkalies—potash, soda, ammonia, or their carbonates, should in all cases be avoided; since the salts which they form with oxalic acid are as poisonous as the acid itself. In the after-treatment (in the stage of collapse) warmth should be applied and stimulants administered.

CHEMICAL ANALYSIS.

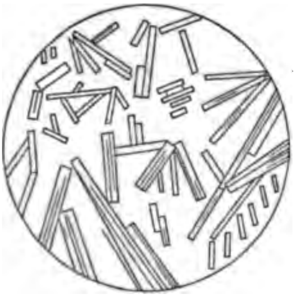
This acid may be met with, either as a solid or in solution in water.

Solid oxalic acid.—It crystallizes in long slender prisms, which, when perfect, are four-sided. (Fig. 9.) In this respect it differs from other common acids, mineral and vegetable. The crystals are unchangeable in air; they are soluble in water and alcohol, forming strongly acid solutions. The solubility in water is variously stated. I have found some specimens much more soluble than others; and the conclusion from the experiments made is, that the acid is soluble in from twelve to fourteen times its weight of water. If there be any adhering nitric acid about the crystals, they are rendered more soluble. It is worthy of remark that this solution, unlike that of some other vegetable acids (tartaric and citric), undergoes no change or decomposition by keeping.

When the crystals are heated on platinum-foil they melt, and are entirely dissipated in a white vapor, without combustion, and without being carbonized. Heated gently in a close tube they melt, and the vapor is condensed as a white crystalline sublimate in a cold part of the tube. The crystals are prismatic, like those obtained from the solution. (See Fig. 10.) There should be no residue whatever if the acid is pure; but the commercial acid generally leaves a slight residue of fixed impurity. By this effect of heat oxalic acid is easily distinguished from those crystalline salts for which it has been sometimes fatally mistaken, namely, the sulphates of magnesia and zinc. These are not volatile, but leave white residues in the form of anhydrous salts. A teaspoonful of oxalic acid in small crystals weighs seventy-six grains, and half an ounce of the crystals is equivalent to three teaspoonfuls.

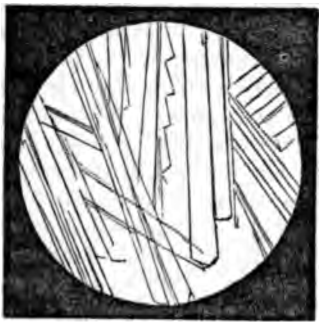
For the further analysis of the acid the crystals may be dissolved in distilled water; but should a suspected *solution* of the poison in water be presented for examination, it will be proper, after testing it with litmus-paper, to evaporate a few drops on a slip of glass, in order to observe whether crystals are obtained. If there should be none, there can be no oxalic acid present. If *long and slender prisms*, having an acid reaction, be procured, then it will be proper to proceed with the analysis of the solution.

FIG. 9.



Crystals of oxalic acid, from a solution, magnified 30 diameters.

FIG. 10.



Crystals of sublimed oxalic acid, magnified 70 diameters.

Tests for the Solution.—1. *Nitrate of silver.* When added to a solution of oxalic acid it produces an abundant white precipitate of oxalate of silver. A solution containing so small a quantity of oxalic acid as not to redden litmus-paper is affected by this test; but when the quantity of poison is small, it would be always advisable to concentrate the liquid by evaporation before applying the test. The oxalate of silver is identified by the following properties: 1. It is completely dissolved by cold nitric acid. If collected on a filter, thoroughly dried, and heated on thin platinum-foil, it is entirely dissipated in a white vapor with a slight detonation. When the oxalate is in small quantity this detonation may be observed in detached particles on burning the filter previously well dried. 2. *Sulphate of lime.* A solution of oxalic acid is precipitated white by lime-water and all the salts of lime. Lime-water is itself objectionable as a test, because it gives a white precipitate with several other acids. The salt of lime which, as a test, is open to the least objection, is the *sulphate.* As this is not a very soluble salt, its solution must be added in rather large quantity to the solution of oxalic acid. A white precipitate of oxalate of lime is slowly formed. This precipitate should possess the following properties: 1. It ought to be immediately dissolved by nitric or hydrochloric acid. 2. It ought not to be dissolved by the oxalic, tartaric, acetic, or any vegetable acid.

Other tests may be used—as, for instance, the chloride of gold, or the sulphate of copper, but they add no force to the evidence afforded by those abovementioned, and we may conclude that when we obtain from an acid solution, a solid acid substance crystallizing in well-defined slender prisms, these crystals remaining unchanged in air, being volatile without combustion, and giving, when dissolved in water, on the addition of nitrate of silver and sulphate of lime, the results above described, there can be no doubt that the substance is oxalic acid. Additional tests may or may not be employed, but any evidence, short of this, should not, it appears to me, be admitted to show the presence of the poison.

In Liquids containing Organic Matter.—The process is the same, whether it is applied to liquids in which the poison is administered, to the *matters vomited*, or lastly, *the contents of the stomach.* Oxalic acid readily combines with albumen and gelatin, and it is not liable to be decomposed or precipitated by these or any other organic substances; it is, therefore, commonly found in solution in the liquid portion, which will then be more or less acid. As a trial-test we may employ either a solution of sulphate of copper or lime-water. 1. A portion of the liquid should be boiled to remove any albumen, and after filtration a solution of sulphate of copper should be added to it. If oxalic acid is present in moderate quantity, a greenish-white precipitate will be formed. 2. Lime-water may be added to another portion of the clear liquid. A white precipitate will be produced, insoluble in acetic acid, if oxalic acid is present.

From milk, gruel, coffee, blood, mucus, and other viscid liquids, oxalic acid is readily separated by the process of dialysis, as described under sulphuric acid (see page 198). The liquid should be first boiled; the coats of the stomach (cut up) being included, if necessary. The distilled water placed on the outside of the tube will receive the acid. This may be concentrated by evaporation. Prismatic crystals may thus be procured, and the silver and lime tests may be applied.

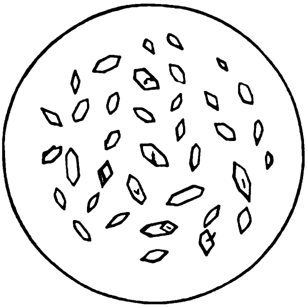
Oxalic acid may be completely separated in a crystalline state from the boiled and filtered organic liquid by the following process: To the filtered liquid, acidulated with acetic acid, *acetate of lead* should be added until there is no further precipitation; and the white precipitate formed, collected and washed. If any oxalic acid was present in the liquid, it would exist in this precipitate under the form of oxalate of lead. To separate oxalic acid from the oxalate of lead we diffuse the precipitate in water, and pass into the liquid, for about half an hour, a current of sulphuretted hydrogen gas, taking care that the gas comes in contact with every portion of the precipitate. Black sulphide of lead will be thrown down; and with it commonly the greater part of the organic matter, which may have been mixed with the oxalate of lead. Filter, to separate the sulphide of lead; the filtered liquid may be clear and highly acid. Concentrate by evaporation; the sulphuretted hydrogen dissolved in the liquid is thereby expelled, and oxalic acid may be ultimately obtained crystallized by slow evaporation in a dial or watch-glass, or on a glass slide for microscopical observation. If there was no oxalic acid in the precipitate, no crystals will be procured by evaporation. If prismatic crystals are obtained, they must be dissolved in water, and tested for oxalic acid in the manner above directed.

If, in the course of the analysis, acetate of lead should give no precipitate with the concentrated liquid, even when neutralized, then oxalic acid is not present in a quantity sufficient to be detected. If it should give a precipitate, still there may be no oxalic acid present. The medical jurist must remember, that the acetate of lead is precipitated by most kinds of organic matter, and by many mineral and vegetable acids and their salts. Thus, if he is operating on the acid contents of a stomach, the presence of Epsom salt (sulphate of magnesia), any alkaline sulphate, common salt (chloride of sodium),—any tartrate, citrate, phosphate, or carbonate, would occasion a white precipitate with the acetate of lead; but these compounds do not yield crystals possessing the properties of oxalic acid. The acetate of lead is not used as a test, but simply as a means of separation.

The presence of oxalic acid in an organic liquid may be detected by another method. Place a portion of the liquid containing the poison in a beaker, and insert in this a tube secured with skin, containing a solution of sulphate of lime. (Fig. 5, p. 198.) By dialysis or osmosis, the oxalic acid will penetrate the membrane, and will form, inside the mouth of the tube, a deposit of crystals of oxalate of lime, known by their octahedral form. (See Fig. 11.)

As oxalic acid is very soluble in alcohol, this liquid may be occasionally employed for separating it from the contents of the stomach and from many organic compounds. Large and perfect crystals may be obtained from the alcoholic solution, and these may be purified and tested by the methods already described.

FIG. 11.



Crystals of oxalate of lime obtained by dialysis of coffee containing oxalic acid, magnified 350 diameters.

In cases of poisoning, the residuary quantity found in the stomach is generally small. In one instance, in which about an ounce and a half had been taken, and the person died in two hours, I found only thirteen grains. This is owing to the early ejection of the greater portion of the acid by vomiting. In a case which occurred at Bristol, in 1868, a woman took three-quarters of an ounce of oxalic acid (360 grains), and died in ten minutes. It is stated that not more than two grains were obtained from the coats of the stomach. The vomiting had been violent, and the greater part of the poison had been thus ejected. It seems that the woman had vomited into a pail containing calcareous water, and it was observed that this water acquired a milky white appearance, owing to the action of the acid on the salts of lime. (*Chem. News*, April 24, 1868, p. 205, and *Pharm. Jour.*, May, 1868, p. 543.) In *Reg.* v. *Cochrane* (Liverpool Summer Assizes, 1857), in which it was charged that two children, aged six and four years respectively, had been wilfully poisoned by their mother, it was stated by the medical witness, Dr. Edwards, that he found forty-two grains of oxalic acid in the stomach of the elder, and twenty grains in that of the younger child. It was not clearly established when or how this large quantity of poison could have been wilfully administered to the children, and the prisoner was acquitted.

The stomach after death may contain no traces of the poison. This will happen when the case is protracted, vomiting has been urgent, or the stomach-pump employed. On the other hand, the poison may be present, but in an insoluble form, when lime or magnesia has been given as an antidote. White chalky masses of *oxalate of lime* may in this case be found adhering to the mucous surface of the stomach, or subsiding as a sediment in the liquid contents. The following process for detecting the acid may be then adopted. The suspected oxalate, previously well washed, should be boiled for about twenty minutes, with an equal weight of pure carbonate of potash. A partial double decomposition takes place; the undissolved residue containing some carbonate of lime, and the liquid some oxalate of potash. The liquid may be filtered, neutralized by diluted nitric acid, and then tested with the tests already described for oxalic acid.

Oxalic acid has not been found in the fluids of the stomach and intestines except in those cases in which it has been taken as a poison. It is not a constituent of any of the secretions of the body. There are two vegetable articles of food which contain it in the form of soluble acid oxalate; namely, common sorrel (*Rumex acetosa*) much used as an esculent herb on the continent, and the garden rhubarb or pie-plant (*Rheum rhaponticum*), the leaf-stalks of which are largely consumed as a substitute for fruit in England. The proportion of acid oxalate in sorrel, is less than an ounce to one gallon of juice. This is so small that it could create no difficulty in the chemical analysis, and unless symptoms of poisoning had existed, no question could be raised to affect the value of the chemical evidence. The presence of the vegetable substance in the stomach, would sufficiently explain the existence of traces of oxalic acid.

The proportion of oxalic acid in the combined state present in the leaf-stalks of edible rhubarb, has not been accurately determined, and it is probably liable to variation according to the stage of growth of the plant. Enormous quantities of it are consumed annually in this metropolis, but we never hear of any accident from its use. The following case, from the *Medical Gazette* (vol. 38, p. 40), is the only instance in which symptoms resembling those of irritant poisoning are stated to have been caused by this vegetable. A family of four persons, after eating very freely of the *leaves* of the domestic rhubarb or pie-plant, boiled and served as "greens," were all of them shortly afterwards seized with severe vomiting. In one of these persons the attack was followed by gastritis; but the others recovered soon after the vomiting. It is stated in the same journal, from an analysis by Dr. Long, that one pound of the plant yielded twenty-four grains of oxalic acid; but as the smallest quantity of acid known to have destroyed life has been sixty grains, it is not probable that any person could eat sufficient to place his life in danger.

On Articles of Clothing.—When there is no other source of evidence, the acid may be detected on cloth, linen, or paper. It does not corrode these substances like a mineral acid, but it slowly produces reddish-brown or orange-colored spots on black cloth without destroying the fibre. Unless the stuff has been washed, the acid remains in the fabric and may there be detected by boiling the stained portion in water. In *Reg.* v. *Morris* (C. C. C., December, 1866), it was proved that the prisoner had attempted to administer a liquid poison forcibly to her daughter, a girl aged six years. It was sour in taste, made her lips smart, and caused vomiting. There was dryness of the lips, and inflammation of the lining membrane of the mouth. No portion of the substance administered could be procured, but a crystalline deposit of oxalic acid was obtained from some stains on the dress of the child. The woman was convicted.

Dr. White (U. S.) has published an elaborate report of a case of poisoning with oxalic acid, in which the symptoms and appearances are contrasted with those caused by disease, and compared with those usually assigned to oxalic acid. The poison was not detected in the contents of the stomach, but the sheets of the bed on which the patient had vomited, yielded from one to two grains of oxalic acid! The patient lived forty hours after vomiting had set in. (*Boston Med. and Surg. Jour.*, Jan. 27, 1870.)

Quantitative Analysis.—The quantity of oxalic acid present in a measured portion of any mixture may be determined by neutralizing the acid, precipitating it entirely with a solution of acetate of lead, and weighing the dry residue thus obtained. The quantity of crystallized oxalic acid may be calculated from the amount of dry oxalate of lead obtained from the liquid. 100 grains of dry oxalate are equivalent to 42 grains of crystallized oxalic acid. To connect weight with measure, it may be stated that a teaspoonful of the small crystals of oxalic acid weighs 76 grains, and three teaspoonfuls correspond to half an ounce (avoirdupois) by weight.

CHAPTER XXVIII.

POISONING WITH TARTARIC ACID—EFFECTS OF ACETIC ACID—VINEGAR—PYROGALLIC ACID—CARBOLIC ACID—OIL OF TAR—CREASOTE—SYMPTOMS AND APPEARANCES PRODUCED BY THESE COMPOUNDS—POISONING WITH PICRIC OR CARBAZOTIC ACID.

TARTARIC ACID.

TARTARIC ACID has been generally considered not to possess any poisonous properties; but one case at least is on record, in which there was no doubt that this acid had acted as an irritant, and destroyed life. The case referred to was the subject of a trial for manslaughter at the Central Criminal Court, in January, 1845 (*Reg.* v. *Watkins*). The accused gave the deceased, a man, æt. 24, by mistake, *one ounce* of tartaric acid instead of aperient salts. The deceased swallowed the whole, dissolved in half a pint of warm water, at a dose; he immediately exclaimed that he was poisoned; he complained of a burning sensation in his throat and stomach, as though he had drunk oil of vitriol, and that he could compare it to nothing but being all on fire. Soda and magnesia were administered with diluent drinks. Vomiting set in, and this symptom continued until death, which took place nine days afterwards. On inspection, nearly the whole of the alimentary canal was found highly inflamed. The accused admitted that he had made a mistake, and tartaric acid was found in the dregs of the cup. The jury acquitted the prisoner.

Another case of poisoning by this acid, with a report of the results of analysis, has been published by M. Devergie. (*Ann. d'Hyg.*, 1851, vol. 2, p. 432.) This case gave rise to a controversy between the late M. Orfila and M. Devergie, the points in dispute relating chiefly to the processes for the detection of the acid in the stomach and tissues. (See *Ann. d'Hyg.*, 1852, vol. 1, pp. 199, 382, and vol. 2, p. 230.)

M. Tardieu describes among the appearances a persistent fluidity of the blood, and, further, that this liquid acquires the color of red currant juice. It communicates this color to the tissues; and in the substance of the organs, especially of the lungs, there are apoplectic effusions. (*Sur l'Empoisonnement*, 1867, p. 253.) There can be no doubt that this acid is absorbed, and that, like oxalic acid, it produces certain changes in the blood. Dragendorff states that it is decomposed in the body, and is eliminated by the urine only in very small quantity.

Dr. Mitscherlich has performed with this acid a series of experiments on animals, which tend to prove that it is not an active poison. He found that while the animal was under its influence its breathing was accelerated, and it then became laborious and slow. Great debility was a prominent symptom, and soon ended in paralysis, death being preceded by slight spasms. He considered this acid to be less noxious than the citric. Half an ounce was given to a small rabbit, and proved fatal in one hour; three drachms killed a similar animal in forty minutes; and two drachms, given to a middle-sized rabbit, produced no effects. In the fatal cases, it was not found to excite inflammation of the small intestines. Tartaric acid appears to enter into the blood, and to act by absorption, for Wöhler detected it as acid tartrate of lime in the urine of animals to which he had administered it. (*Med. Times*, Sept. 1845, p. 341.) Sir R. Christison states that he has given to cats one drachm of this acid in solution, without apparently producing any inconvenience to the animal! and that a surgeon of his acquaintance had known six drachms of tartaric acid to have been taken by an adult, in mistake for carbonate of potash, without exciting unpleasant symptoms. (*On Poisons*, p. 227.)

Treatment.—The same as in poisoning by oxalic acid.

Chemical Analysis.—Tartaric acid in powder is known by the following characters: 1. When heated on platinum-foil it burns with a pale reddish-colored flame evolving a peculiar odor and leaving an abundant residue of carbon. 2. It forms an acid solution in water, which when moderately concentrated yields a granular precipitate with a few drops of caustic potash slowly added (bitartrate). A little alcohol facilitates the precipitation. 3. When a few drops of the acid solution are evaporated on glass, it crystallizes in an irregular plumose form. 4. The solution is precipitated white by lime-water, when the latter is added in large quantity; the precipitate being immediately dissolved by a slight excess of the acid. 5. It gives no precipitate, or only a slight opacity with nitrate of silver (thus known from oxalic acid). 6. It is not precipitated by chloride of calcium. 7. When exactly neutralized by potash, and nitrate of silver is added, a white precipitate is formed, which is immediately blackened and reduced to the state of metallic silver on heating the liquid to 212°. 8. When the powdered acid is heated with strong sulphuric acid, it is blackened.

Organic Mixtures.—If the acid be not discovered in the stomach in the state of powder or crystals, we may obtain it by digesting the contents in alcohol, in which this vegetable acid is quite soluble.

ACETIC ACID.

This acid has been generally excluded from the class of poisons. Common *vinegar*, which contains only five per cent. of acetic acid, has been often taken in large doses without injurious consequences. From the experiments performed by Orfila on dogs, and from one case which he reports as having occurred in the human subject, acetic acid, when concentrated, appears to exert an irritant action on the body. (*Annales d'Hygiène*, 1831, vol. 2, p. 159; also *Toxicologie*, vol. 2, p. 198.) This is not more than we might have expected, seeing that the concentrated acid is highly corrosive. In the case referred to, the deceased, a girl, æt. 19, was found dying on the highway. She suffered from convulsions, complained of pain in the stomach, and died in a short time. On inspection, the stomach was found neither softened nor corroded, but its mucous membrane near the pylorus was almost black. The mucous glands were prominent, and the vessels were filled with dark coagulated blood. There can be no doubt that the glacial acetic acid, from its well-known solvent action on animal substances, would operate as a corrosive poison and destroy life. A fatal case of poisoning with this liquid occurred at Plumstead in August, 1873. A woman gave by mistake to her child, æt. 2 years, a dose of glacial acetic acid which had been used for removing warts. The child suffered the most intense pain, and died in about thirty-six hours. No post-mortem examination was required for the coroner's inquest!

These remarks equally apply to *aromatic acetic acid*, which contains ninety per cent. of glacial acetic acid, combined with some aromatic oils. In one case in which this was incautiously used, it produced corrosion and inflammation of the lining membrane of the nostrils and soft palate. The *treatment* would consist in the free administration of milk and carbonate of soda.

Vinegar, which may be regarded as an organic mixture containing a small proportion of acetic acid, may be examined by distilling a portion, and testing the distilled liquid for the acid. Vinegar, as it exists in commerce, always contains a small quantity of sulphuric acid, and occasionally traces of arsenic, lead, and copper. In general it is easily recognized by its odor. Pelletan observed, in the case of a child, that the abuse of vinegar led to a thinning of the mucous membrane of the stomach; and Landerer remarked that the milk of a wet-nurse who had been in the habit of taking large quantities of the vinegar of roses, became thin, very acid, and deficient in casein and oil. The infant which she was suckling gradually wasted and died, and the woman herself suffered severely. (Heller's *Archiv.*, 1847, 2 H. S 185.)

Analysis.—Acetic acid, if in the free state, would be perceptible by its odor. It may be separated from the contents of the stomach by distillation. It forms a dry crystallizable salt with soda from which strong acetic acid may be obtained by heating it with sulphuric acid.

PYROGALLIC ACID.

Poisonous properties have been attributed to this well-known substance, which is so much employed in photography. I have not met with any case of poisoning by it in the human subject; but according to M. Personne, it operates powerfully on animals. Two healthy dogs were selected, and into the stomach of one a dose of two grains of pure pyrogallic acid, dissolved in water, was injected; and twice this quantity was administered to the other dog. The animals died after fifty and sixty hours respectively. The symptoms are said to have resembled those of phosphorus-poisoning, and after death the muscular tissue of the heart was found in each case to have undergone fatty degeneration! The acid is supposed to act like phosphorus in arresting oxidation changes by absorbing and removing oxygen. The alkalinity of the blood would favor this chemical action. (*Medical Press*, December, 1869; *Amer. Jour. of Med. Sci.*, July, 1870, p. 275.)

CARBOLIC ACID—PHENIC ACID—OIL OF TAR—CREASOTE.

The oil of tar is a powerful vegetable irritant. In 1832, about ten drachms of it caused the death of a gentleman, to whom it had been sent by mistake for a black draught. The druggist who sent it was tried for manslaughter, but acquitted. Its irritant properties are owing to creasote, carbolic acid, and other compounds. Of these the most important in a practical point of view is carbolic acid, which has come into considerable use of late years as a disinfectant.

Carbolic acid is a crystalline product of the fractional distillation of the oil of tar. In an impure state it is a black tarry-looking liquid, and has been long known as impure creasote. The crystals of pure carbolic acid melt at 95°, and the oily-looking liquid boils and is entirely volatilized at 370°. It is sold commercially in a liquid form. Many instances of poisoning by this substance are now on record, the greater number having arisen from accident. Five deaths are recorded to have taken place from it in four years (1863–7). It has such a powerful odor and taste that it could not be easily administered with homicidal intent. In a concentrated form it has a strong local action, and is a corrosive irritant, but it also affects the brain like a narcotic poison. It acts on the unbroken skin, whitens it, hardens it, and destroys its sensibility for some time. It acts in a similar way on the mucous membrane, whitening, hardening, and corrugating it. In three instances it is reported to have destroyed life as the result of external application. (*Brit. Med. Journal*, Oct. 8, 1870.) In one case the person died in two hours. The acid had been rubbed into the skin to cure the itch. (*Pharm. Jour.*, March 22, 1878.) A girl under five years of age died from the absorption of this poison. An incision had been made in the arm in a surgical operation. The wound was covered with lint, soaked in carbolic acid, but without actual contact. In one hour the child was found insensible, and the face livid. She passed into a state of complete coma, and died half an hour later. (*Amer. Jour. Med. Sci.*, July, 1873, p. 280; also *Lancet*, June 7, 1873.)

The acid has a powerful *local action*. A girl, æt. 13, had a splinter beneath her nail. This was removed, and the tip of the finger was dipped into a bottle half full of carbolic acid. There was no pain. A linen compress saturated with the acid was tied round the finger. On the following day this portion of the finger was of a gray color, and completely insensible. The part mortified, and on the fifteenth day the finger was removed. It was dry, horny, and mummified. (Bouchardat, *Ann. de Thérapeut.*, 1874, p. 214). A child, æt. 4½, had an operation performed on the arm, requiring an incision four inches long. The wound was covered with lint soaked in pure carbolic acid. In spite of the attempt to prevent direct contact, the acid had penetrated into the wound and had been absorbed. Symptoms of poisoning came on, followed by complete coma and death in half an hour. (*Amer. Jour. Med. Sci.*, July, 1873, p. 279.) It thus operated as a cerebral poison.

Symptoms and Appearances.—When the poison is swallowed in solution in a moderately concentrated state, the patient experiences a hot burning sensation, extending from the mouth to the stomach. The symptoms come on in the act of swallowing; the lining membrane of the mouth is whitened and hardened. There is severe pain in the stomach, with vomiting of a frothy mucus. The skin is cold and clammy, the lips, eyelids, and ears are livid; the pulse 120 and intermittent; breathing difficult, with frothing at the mouth. There is insensibility, which comes on speedily, and rapidly passes into coma with stertorous breathing; a strong odor of carbolic acid in the breath and in the room; the pupils are contracted and insensible to light. The fæces and urine, when passed, have been dark-colored. These symptoms show that carbolic acid is really a cerebral poison.

Among the *appearances* after death the following have been observed: the interior of the mouth and jaws whitened, sometimes corroded; the œsophagus also is white, hard, and corrugated. The coats of the stomach have presented a horny consistency, without any signs of inflammation. The lungs have been found gorged with dark-colored blood, and the bronchia filled with a brown-red thick mucus.

I. M., æt. 32, swallowed by mistake a solution of the acid in water which had been prepared as a disinfectant. He was almost immediately attacked with sickness, cold sweats, stupor, and insensibility.

These symptoms were followed by coma, a general loss of sensibility, and paralysis of all reflex movements. The cornea and conjunctiva were insensible, and the pupils much contracted; the breathing frequent and stertorous; the pulse small and quick (120). No urine had passed, but some which was drawn off by a catheter had a purple tint, and smelt strongly of the acid. The man was bled. The blood was thick, of a dark-brown color, and this also had a strong odor of the acid. Owing to the paralyzed state of the pharynx he was unable to swallow. The man died on the same day, apparently asphyxiated. (Bouchardat, *Ann. de Thérap.*, 1873, p. 97; also 1874, p. 215.)

In October, 1867, a child, under two years of age, swallowed about two teaspoonfuls of the ordinary brown liquid sold as carbolic acid. She was brought to Guy's Hospital, and was seen ten minutes after swallowing the poison. She was quite insensible; the pupils were contracted and insensible to light; the conjunctiva insensible; pulse 120; skin cold and clammy; lips blue; respiration much impeded. There was a strong tarry odor in the breath. The child had vomited a little frothy fluid. She had lost the power of swallowing. Tracheotomy was performed to relieve the breathing. The child died twelve hours after taking the poison. It vomited, a few hours before death, a quantity of liquid smelling strongly of carbolic acid, and at this time the pupils became sensitive to light.

On inspection, the lining membrane of the mouth, fauces, and œsophagus was white and dense; it was easily detached. There were patches of redness about the stomach, chiefly on the rugæ. The bronchi contained a brown-red thick mucus, which choked the tubes; it contained blood, and smelt strongly of carbolic acid. Its surface was pinkish-red, and there were patches of thin false membrane adhering to it. The lungs were gorged with blood, and had a tarry odor. The cause of death appears to have been chiefly the injury done to the lungs and air-passages. (*Guy's Hosp. Rep.*, 1868, p. 234.) Other fatal cases, with the symptoms and appearances resembling the above, will be found in the *Lancet* for 1873 (vol. 1, pp. 302, 816.) In the same volume, p. 821, a case is reported in which the diluted acid was injected by mistake into the vagina. It excoriated all the surrounding parts.

Fatal Dose.—A woman died from swallowing a wineglassful of carbolic acid, probably a weak aqueous solution. She did not speak after taking it, and died in about *half an hour*. (*Pharm. Jour.*, July, 1872, p. 75.) In 1867 a child, under two years, died in twelve hours from two teaspoonfuls of the ordinary brown liquid carbolic acid. (*Guy's Hospital Reports*, 1867, p. 233.) In another case a tablespoonful killed a young man. In a case which occurred to Mr. Jeffreys, an adult died in fifty minutes after taking from one to two tablespoonfuls of the liquid acid. (See Husemann's *Jahresbericht*, 1872, p. 523.) A case is reported in the *Lancet* (1873, p. 302) in which death took place in less than an hour.

Treatment.—In spite of severe symptoms there may be recovery. A man drank by mistake between two and three drachms of the acid. He immediately fell in an insensible state and was convulsed. He was seen by a medical man in eighteen minutes. He appeared then to be moribund. The extremities were cold, the pulse was scarcely perceptible, the heart's action irregular, the breathing stertorous, he was quite insensible and was in a state of intense trismus (lockjaw). The contents of the stomach were drawn off. The man rapidly recovered, but for some days he suffered from irritation of the throat and gastritis. (*Amer. Jour. Med. Sci.*, 1873, p. 566.) The recovery in this case was entirely due to the early removal of the poison from the stomach by the stomach-pump. This is the best plan of treatment, and as the patient is usually insensible, it is easily carried out. The stomach should be well washed out with tepid water until the smell has disappeared.

Analysis.—The strong and peculiar odor perceptible in the breath, in the vomited matters, and in the room, generally suffice to indicate the nature of the poison. Carbolic acid is partially dissolved by water, and is very soluble in alcohol, ether, or solution of potash. It has no acid reaction, but it gives a greasy stain to paper, and burns with a smoky flame. The watery solution is slightly acid. There is no test for its presence so delicate as the odor. It may be separated from the contents of the stomach by washing them with ether, decanting the ethereal liquid, and allowing the ether to evaporate. Oily-looking globules are thus obtained, having the peculiar odor of carbolic acid, and more or less brownish colored. On mixing these with water, and adding a persalt of iron, the liquid acquires a dark purple or inky color. Bromine gives a white percipitate with a weak aqueous solution readily soluble in an excess of carbolic acid.

The poison has been detected in the urine by distilling this liquid with sulphuric acid. It gives to the urine a dark color, and at the same time imparts to it its peculiar odor. Hoffman detected it in the urine of dogs and cats which had been killed by carbolic acid in from twenty-four to sixty hours. (Dragendorff, *Man. de Tox.*, 1873, p. 515.)

Coralline.—There is a derivative of carbolic or phenic acid, under the name of coralline, to which MM. Tardieu and Roussin assign poisonous properties. As a dye-stuff it constitutes a large branch of manufacture; it is used for dyeing silk and woollen of a red color, as also for paperstaining. By oxidation the phenic is converted into rosolic acid, and this acid, by combination with ammonia, produces the red compound called coralline. The yellow coralline is simply a modification of rosolic acid. Arsenic does not enter into the composition of either.

These dyes, when used for socks and stockings, have been observed to cause a vesicular eruption on the skin. This local action has been followed by some constitutional disturbance. (*Ann. d'Hyg.*, 1869, vol. 1, p. 262.) MM. Tardieu and Roussin found that the coralline dye could be extracted by alcohol, and further that a dog and a rabbit were killed by the injection of a portion of it under the skin. The dyed articles were said to be of English manufacture. These effects were clearly due to the local irritation of the skin set up by this noxious dye.

Anilin red has also been used for dyeing silk and woollen, but in this case, the effects have been traced to the action of arsenic left as an impurity in the colored compound. Coralline red is insoluble in cold

These symptoms were followed by coma, a general loss of sensibility, and paralysis of all reflex movements. The cornea and conjunctiva were insensible, and the pupils much contracted; the breathing frequent and stertorous; the pulse small and quick (120). No urine had passed, but some which was drawn off by a catheter had a purple tint, and smelt strongly of the acid. The man was bled. The blood was thick, of a dark-brown color, and this also had a strong odor of the acid. Owing to the paralyzed state of the pharynx he was unable to swallow. The man died on the same day, apparently asphyxiated. (Bouchardat, *Ann. de Thérap.*, 1873, p. 97; also 1874, p. 215.)

In October, 1867, a child, under two years of age, swallowed about two teaspoonfuls of the ordinary brown liquid sold as carbolic acid. She was brought to Guy's Hospital, and was seen ten minutes after swallowing the poison. She was quite insensible; the pupils were contracted and insensible to light; the conjunctiva insensible; pulse 120; skin cold and clammy; lips blue; respiration much impeded. There was a strong tarry odor in the breath. The child had vomited a little frothy fluid. She had lost the power of swallowing. Tracheotomy was performed to relieve the breathing. The child died twelve hours after taking the poison. It vomited, a few hours before death, a quantity of liquid smelling strongly of carbolic acid, and at this time the pupils became sensitive to light.

On inspection, the lining membrane of the mouth, fauces, and œsophagus was white and dense; it was easily detached. There were patches of redness about the stomach, chiefly on the rugæ. The bronchi contained a brown-red thick mucus, which choked the tubes; it contained blood, and smelt strongly of carbolic acid. Its surface was pinkish-red, and there were patches of thin false membrane adhering to it. The lungs were gorged with blood, and had a tarry odor. The cause of death appears to have been chiefly the injury done to the lungs and air-passages. (*Guy's Hosp. Rep.*, 1868, p. 234.) Other fatal cases, with the symptoms and appearances resembling the above, will be found in the *Lancet* for 1873 (vol. 1, pp. 302, 816.) In the same volume, p. 821, a case is reported in which the diluted acid was injected by mistake into the vagina. It excoriated all the surrounding parts.

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Anilin red has also been used for dyeing silk and woollen, but in this case, the effects have been traced to the action of arsenic left as an impurity in the colored compound. Coralline red is insoluble in cold water. It is very soluble in boiling alcohol. The color is not changed by alkalies. Anilin red is very soluble in cold water; the color is destroyed by ammonia, but reappears on the addition of an acid.

Public attention has been directed to the noxious effects produced by these dyes when employed for articles of clothing worn next to the skin, and there is reason to believe that the practice has been discontinued in this country.

PICRIC OR CARBAZOTIC ACID.

This is a solid crystalline acid, usually seen in yellow prisms, having an intensely colorific power. In the first edition of this work it was briefly noticed as a poison operating as a narcotico-irritant on animals, *i. e.*, affecting the brain and spinal marrow. Ten grains sufficed to kill a dog in less than two hours. The prominent symptoms were tremor of the limbs, stupor, and convulsions. After death the inner coat of the stomach and intestines, the muscles, skin, and the bloodvessels throughout the body were found to be more or less dyed of an intensely yellow color. This staining of the tissues furnished a clear proof that the acid had been carried into the blood by absorption. The late Dr. Calvert proposed to utilize this property by causing the acid to be mixed in small proportion with arsenic and other colorless poisons. The staining of the skin and conjunctiva during life and the yellow coloring given to the muscles and viscera in the event of death, would, it was supposed, reveal the fact of poisoning. Independently of color, it possesses an intensely bitter taste, and thus would impart a strong taste to tasteless poisons. As the substance was itself a poison, the suggestion was never carried into practice.

From its intense bitterness, it is said to have been at one time employed in the brewing of ale as a substitute for hops.

In cases of poisoning with this acid, the urine which is first passed is of a yellow color, showing that the acid is eliminated by the kidneys.

Analysis.—The bitter taste and yellow color are sufficient to identify picric acid. It may be separated from organic substances by boiling them in strong alcohol acidulated with hydrochloric acid. This liquid may be concentrated by evaporation. A slip of flannel or silk is dyed of an intense yellow color when immersed in the acid decoction. The acid may be separated from beer by a similar process, substituting sulphuric for hydrochloric acid. (Dragendorff, *Man. de Toxicol.*, 1873, p. 510.) It has been stated that this substance, when used as a yellow or orange dye for woollen socks and stockings, has given rise to an eczematous eruption on the skin. Although it possesses locally irritant properties, it is probable that the yellow and orange dyes referred to have been really derived from phenic or carbolic acid.

ALKALINE POISONS.

CHAPTER XXIX.

POISONING BY THE ALKALIES—POTASH, SODA, AND THEIR CARBONATES—SYMPTOMS—APPEARANCES—TREATMENT—ANALYSIS—AMMONIA AND CARBONATE OF AMMONIA (SAL VOLATILE)—FATAL ACTION ON THE LUNGS—CHEMICAL ANALYSIS OF AMMONIA.

Alkalies.—The alkaline poisons are few in number; but the saline combinations which they form are very numerous. A selection has been made of the most important of these compounds, in a toxicological point of view, and these will be considered after the alkalies. By an alkali we here understand a substance soluble in water and alcohol. Its aqueous solution has a soapy feel. It corrodes and dissolves many organic substances. It neutralizes acids and forms salts. Litmus-paper reddened by an acid is rendered again blue by an alkali. The most delicate test of alkalinity in a liquid is the arsenio-nitrate of silver (made by mixing equal parts of saturated solutions of arsenious acid and nitrate of silver). It produces in a solution containing a very small portion of alkali, a dense yellow precipitate (arsenite of silver). The alkaline poisons which will be first considered are Potash and Soda.

POTASH AND SODA.

Symptoms.—The symptoms produced by potash and soda, when taken in large doses, are so similar, that one description will serve for both. It must be observed, that cases of alkaline poisoning are extremely rare, and have been hitherto chiefly the result of accident. The most common form in which these poisons are met with, is in the state of pearl ash (carbonate of potash) and soap-lees (carbonate of potash or soda mixed with caustic alkali). The patient experiences, during the act of swallowing, an acrid, caustic taste—the alkaline liquid, if sufficiently concentrated, softens and corrodes the lining membrane of the mouth. There is a sensation of burning heat in the throat, extending down the gullet to the pit of the stomach. Vomiting is not always observed; but when it does occur, the vomited matters are sometimes mixed with blood of a dark-brown color; and with detached portions of mucous membrane—this effect depending on the degree of causticity in the liquid swallowed. The skin is cold and clammy; there is purging, with severe pain in the abdomen, resembling colic. The pulse is quick and feeble. In the course of a short time, the lips, tongue, and throat become swollen, soft, and red.

Appearances after Death.—In recent cases there are strong marks of the local action of the poison on the mucous membrane of the mouth, throat, and gullet. This membrane has been found softened, detached, and inflamed, in patches of a deep chocolate color,—sometimes almost black. The same appearance has been met with in the lining membrane of the larynx and trachea. The stomach has had its mucous surface eroded in patches, and there has been partial inflammation. In one instance, as the result of the action of soda, I found it puckered and blackened.

Dr. Barclay has reported a case of chronic poisoning by potash, which furnishes a good illustration of the after-effects and appearances caused by this poison. A woman, aged 44, was admitted into St. George's Hospital, about six hours and a half after she had swallowed a quantity of American potash,—probably a saturated solution of carbonate of potash (American pearl ash). She had vomited immediately after taking it. The mouth and fauces were much corroded. There was burning pain in the throat and gullet, extending downwards to the stomach; but there was no tenderness on pressure. Two days after her admission, there was a little vomiting. The mucous membrane, so far as it could be seen, was destroyed; there was some difficulty of swallowing, and occasionally pain after food had entered the stomach. In about a month there was frequent vomiting, with pain on pressure, and constipation; when food or medicine was taken, there was much pain in the stomach, and in a short time the food was ejected. As the case progressed, nothing could be retained on the stomach, and shortly before death the patient was supported only by nutritive injections. She died from starvation on July 8, about *two months* after taking the alkali. On inspection, the lower part of the gullet was found much contracted, the lining membrane entirely destroyed, and the muscular coat exposed. The external coats were much thickened. The cardiac orifice of the stomach, where the ulceration ceased, was considerably contracted. At the pyloric end, the mucous lining presented a large and dense cicatrix, obstructing all communication with the duodenum except by an orifice no larger than a probe. The intervening portion of the stomach was healthy, as were also the large and small intestines. (*Med. Times and Gazette*, Nov. 26, 1853, p. 554.) Orfila refers to two cases of poisoning by carbonate of potash, in each of which, half an ounce of this substance was taken by mistake for aperient salts. The patients,—two young men, recovered from the first effects, but ultimately died; the one three months, and the other four months, after the poison had been taken.

The secondary fatal effects of these poisons appear to be due to constant purging, great irritability of the stomach, leading to incessant vomiting, or loss of the functions of this organ from the destruction of the lining membrane, and stricture either of the gullet or of the apertures of the stomach—any of which causes may destroy life at almost any period. A fatal case of stricture, produced by soap-lees, after the lapse of two years and three months, is reported by Dr. Basham (*Lancet*, March 2, 1850). The constant use of the alkalies or their carbonates appears to be productive of latent mischief; yet the quantity which may be sometimes taken in divided doses without destroying life is enormous. Dr. Tunstall, of Bath, relates the case of a man who for eighteen years had been in the habit of taking bicarbonate of soda to remove dyspepsia. It is stated that for sixteen years he took *two ounces* of the bicarbonate daily! The man died suddenly, and on examining the stomach it was found to be greatly distended and extensively diseased—conditions which were referred by Dr. Tunstall to the action of the bicarbonate of soda. (*Med. Times*, Nov. 30, 1850, p. 564.)

In a case which occurred to Dr. Deutsch (*Berlin Med. Zeitung*, 1857, No. 51), a man, æt. 55, drank by mistake a quantity of *soap-lees*, containing about 30 per cent. of caustic potash. It was calculated that the quantity taken must have contained half an ounce of potash, of which a fourth part was considered to have reached the stomach. He was seen almost immediately. The lining membrane of the mouth and throat had a bluish-red color, was separating in shreds, and easily bled on being touched. The man complained of an insupportable burning pain extending from the mouth to the stomach, a nauseous taste and a constriction in the gullet. He could not swallow; any attempt at swallowing gave rise to constriction of the throat. Choking and an inclination to vomit existed, but he did not completely vomit. There was cold perspiration, with paleness of the face—collapsed features, slight convulsions—hiccough, and a rapid, small, thready pulse. The abdomen was distended and tender to the touch. The taking of liquids produced vomiting of bloody shreds of mucous membrane. In eight days the inflammation was reduced. After six weeks there was still difficulty of swallowing; this increased, and the man died from starvation twenty-eight weeks after he had swallowed the alkali. On inspection, the throat and upper part of the gullet presented nothing abnormal; but the gullet became thicker and more contracted as it descended to the stomach, so that the opening into the stomach would scarcely admit a crow-quill. This was owing to a thickening of the mucous membrane. The stomach was empty, small, contracted, and bloodless, but free from any organic changes. (*Med. Times and Gaz.*, May 22, 1857, p. 597.)

The following case was admitted into Guy's Hospital in September, 1857. A child, under two years of age, had drunk from a cup, about an hour before, a mouthful of *soap-lees*. In the course of an hour or two difficulty of breathing came on with intense heat of skin. The child died twelve hours after swallowing the fluid. The mouth, tongue, and œsophagus had a yellowish-brown hue, and the lower part of the latter was of a dark-brown color. The stomach was contracted, and the mucous membrane was of a pink hue from injection. Near the pyloric end the rugæ were of a dark-brown color, a chemical result of the action of the alkali. The mucous membranes were hard and had a horny feel. The parts about the larynx were much swollen. (*Guy's Hosp. Reports*, 1859, p. 133.)

Stricture of the œsophagus is one of the results of this form of poisoning, and the patient may die at a long period after taking the poison. Dr. Ashhurst, U. S., describes the case of a girl, æt. 4, who died from this cause, *two years* after swallowing an alkaline liquid. The œsophagus was found contracted to three-eighths of an inch, or to one-third of its circumference, towards the lower part. (*Amer. Jour. Med. Sci.*, April, 1871, p. 394.)

Fatal Dose and Period of Death.—The most rapidly fatal case which I have found reported, is that of a boy, who died in *three hours* after swallowing three ounces of a strong solution of carbonate of potash. In another case, a child, aged 3 years, took a small quantity of a concentrated solution of pearl ash, which had deliquesced, and died in twenty-four hours. Death was caused in this instance by the inflammation induced in the larynx causing an obstruction to breathing. In this respect the caustic alkalies may destroy life like the mineral acids, by the local effects on the air-passages. In an instance which was communicated to me by a pupil, a lady swallowed, by mistake, one ounce and a half of the common solution of potash of the shops, which contains about five per cent. of caustic alkali. She recovered from the first symptoms of irritation, but died seven weeks afterwards, from pure exhaustion, becoming greatly emaciated before her death. The alkali had probably destroyed the lining membrane of the stomach, and had thus impaired digestion.

Treatment.—We may administer freely, water containing acetic or citric acid dissolved—lemon-juice, or the juice of oranges. Demulcent drinks, as albumen, milk, gruel, or barley-water, will also be found serviceable. The free exhibition of oil has also been found useful.

Chemical Analysis.—Caustic potash and soda are known from their respective carbonates by giving a brown precipitate with a solution of nitrate of silver. The *carbonates*, on the other hand, yield a whitish-yellow precipitate and effervesce with acids. Caustic *potash* is known from caustic *soda* by the following characters: 1. Its solution, when not too much diluted with water, is precipitated of a canary-yellow color by chloride of platinum. 2. It is precipitated in granular white crystals, on the addition of an excess of a strong solution of tartaric acid. This test answers better by adding the alkali gradually to the acid, and by the addition of a little alcohol to the mixture. Caustic *soda* is not precipitated by either of these tests, which will serve equally to distinguish the salts of potash from those of soda, if we except the binoxalate and bitartrate of potash, which, from being but little soluble in water, are not precipitated. 3. If we neutralize the two alkalies by diluted nitric acid, and crystallize the liquids on a slip of glass, should the alkali be potash, the crystals will be in the form of long slender fluted prisms; if soda, of rhombic plates. (See Figs. 6 and 7, page 211, *ante*.) 4. A fine platinum wire may be dipped into the alkaline liquid, and then dried by holding it above the flame of a spirit-lamp. In this way a thin film of solid alkali is obtained on the wire. On introducing this into the colorless part of the flame, if it be potash, the flame will acquire a lilac color; if soda, a rich yellow color. The test applies to the salts of the two alkalies. Care must be taken that the platinum wire is perfectly clean. When the quantity of alkali or alkaline salt is large, the experiment may be performed in a platinum capsule, alcohol being added to the salt, and the mixture boiled.

The *carbonates* of potash are known from those of soda by the above tests. The *carbonate* is known from the *bicarbonate* of either alkali, by the fact that the former yields immediately a white precipitate, with a solution of sulphate of magnesia, while the latter is unaffected by this test until the mixture is heated. It is important for the analyst to remember that caustic potash and soda, their respective carbonates, and the sesquicarbonate of ammonia, are often contaminated with oxide of lead, and give a black precipitate with sulphuretted hydrogen or hydrosulphuret of ammonia. This happens whenever the solutions of these salts have been kept for some time in flint-glass bottles.

In Liquids containing Organic Matter.—Such liquids are frothy; they possess an alkaline reaction, a peculiar alkaline odor, and are unctuous to the feel. Potash and soda soften and dissolve most kinds of animal and vegetable matter. They also act upon woollen articles of clothing. If the organic liquid be highly alkaline, and gives out no odor of ammonia, either by itself or on distilling a portion of it with caustic potash, the alkali may be either *potash* or *soda*, or their *carbonates*. The latter would be known by the liquid effervescing on adding a portion of it to an acid. The organic liquid may be evaporated to dryness, then heated to char the animal and vegetable matter, and the alkali will be recovered from it in the state of carbonate by digesting the incinerated residue in pure alcohol. It has been also recommended to neutralize by muriatic acid, to evaporate, incinerate, and procure the alkali for analysis in the state of chloride. Mere traces of these alkalies furnish no evidence of poisoning, since all the animal liquids and solids yield soda, and many of them potash. In no case will the discovery of the alkalies be any proof of poisoning, unless the alkali be in *large* quantity, and the marks of its action be apparent in the throat and stomach.

If the alkali be *ammonia*, this will be announced by the odor, and it may then be obtained by distillation. If it be in small proportion, this can afford no evidence of poisoning; since many animal fluids contain ammonia, and in those which do not contain it, it is easily generated either by spontaneous decomposition, or sometimes even by the heat required for distillation with potash. Should the alkali be in large quantity, this is no evidence of poisoning by it, unless we, at the same time, discover obvious marks of its local action on the mouth, throat, gullet, and stomach.

According to the experiments of Orfila, potash is *absorbed* and conveyed into the blood. The alkali is eliminated by the urine, which is thereby rendered alkaline. When he gave about one drachm of potash to dogs, the presence of this alkali was detected after the lapse of six hours in the liver, spleen, and kidneys. Owing to the solvent action of this poison on fibrin and albumen, the blood, although it may be darker in color, is never found coagulated in the vessels after death.

AMMONIA.

Vapor.—The vapor of strong ammonia is poisonous. It may destroy life by producing violent inflammation of the larynx and of the lungs. The vapor produces a feeling of choking, with a sense of great heat in the throat; it appears to suspend the power of breathing, and the pain and heat in the throat remain for a long time. The vapor is often most injudiciously employed to rouse a person from a fit. A case is on record of an epileptic having died, under all the symptoms of croup, two days after the application of the vapor of strong ammonia to his nostrils. A singular case of recovery from the poisonous effects of this vapor, by Dr. Souchard, will be found in the *Annales d'Hygiène* (1841, vol. 1, p. 219).

In another case in which the vapor was breathed accidentally, in the manufacture of ice from liquefied ammonia, is reported (Husemann's *Jahresbericht*, 1872, p. 1070); the effects were chiefly manifested on the lungs, and many days elapsed before recovery took place.

Symptoms and Appearances.—A strong *solution* of ammonia, when swallowed, produces symptoms similar to those described in speaking of potash. The chief difference observed is, that the sense of heat and burning pain in the throat and stomach are much greater. Cases of this form of poisoning are rare. Dr. Souchard relates an instance which occurred in France, where a boy, only 6 years old, poisoned his younger sister by pouring several teaspoonfuls of strong solution of ammonia down her throat. In the following instance of poisoning by ammonia, the exact quantity taken was unknown, but the solution was sufficiently strong to act chemically on the mouth. A woman, æt. 24, swallowed about half a wineglassful of a mixture containing a large quantity of strong ammonia, put into it by mistake. She immediately fell backwards in a state of insensibility, and appeared as if choked. When seen about six hours after the accident, she complained of severe burning pain down her throat and in the stomach, which was tender on pressure. There was great debility, the voice was reduced to a whisper, and the countenance expressed anxiety. There was also great difficulty of swallowing, the pupils were widely dilated, the breathing was difficult, the tongue coated with a white fur, painful, and tender; two or three patches of its mucous membrane had peeled off, and there were convulsive twitches of the right arm. Diluted vinegar and other remedies were employed, but the patient did not entirely recover from the effects until after the lapse of ten days. On the fifth day there were still great pain and tenderness in the abdomen. (Case by Mr. Wilkins, *Lancet*, April 4, p. 385.) A case is referred to in the *Journal de Pharmacie* (Oct. 1846, p. 285), in which from one to two drachms of ammonia, unknowingly administered, caused death. There was violent vomiting, with bloody purging; and, on inspection, blood was found effused in the intestines. There was also a remarkably fluid state of the blood in the body. In another instance, a man walked into a druggist's shop, and asked for a small quantity of ammonia to take some spots out of his clothes. The druggist poured about a teaspoonful and a half into a glass. The man suddenly swallowed it, and fell instantly to the ground. He soon afterwards died, complaining of the most excruciating pain. (*Journal de Chimie Médicale*, 1845, p. 531.)

A similar case occurred at Halifax in April, 1857; a man swallowed a large dose of ammonia, and died in a quarter of an hour. In one instance a strong dose of the solution killed a man in *four minutes*, by causing suffocation. (*Christison*, p. 167.) In other cases, in spite of a large dose, death has taken place slowly.

Dr. Potain met with an instance in which a man swallowed upwards of three ounces of the commercial solution of ammonia, and he did not die from the effects until the eleventh day. (*Journal de Chimie Médicale*, 1862, pp. 311 and 474.)

Dr. Stevenson reports the following case of poisoning with solution of ammonia: H. H. was admitted into Guy's Hospital in July, 1871. He had shortly before drunk a teaspoonful of strong liquor ammoniæ (s. g. .88). The lips, tongue, tonsils, and uvula were much swollen, red, and glazed, and covered here and there with pieces of white epithelium. He had great difficulty of breathing, complained of slight pain in the abdomen, turned over on his side, became blue in the face, and expired immediately without any struggle for breath. On inspection, the mucous membrane of the mouth and pharynx were found to be red and glazed. The œsophagus was intensely red throughout, and at the lower part was of a dark purple color, which ceased abruptly at the stomach. There was much œdema about the larynx, and the mucous membrane of the trachea and bronchi was thickened and injected. Both lungs were œdematous and gorged with blood. The mucous membrane of the stomach presented a patch of injection about four inches in diameter, and it was here thin. The ammonia had probably come in contact with this part of the stomach; elsewhere it was thick, pale, and coated with slimy mucus. Both sides of the heart contained dark fluid blood. (*Guy's Hosp. Rep.*, 1872, p. 225.) It is probable that in this case the alkali caused death by its effects on the respiratory organs.

In August, 1854, a woman swallowed ten drachms of a solution of ammonia (strength not stated); she threw the glass from her and rushed into an adjoining room. When seen by a medical man she was in a sitting position, having on her knees a basin containing a large quantity of stringy salivary fluid, with a few streaks of blood. Her face was pale, the eyes were haggard and injected, the lips presented much swelling, and also redness, which extended to the mouth and throat. There was entire loss of voice. There was pain in the pharynx and stomach. The pulse was slow, and the limbs were cold. Some spoonfuls of vinegar were given, but were swallowed with difficulty. The pain in the stomach was severe, and was increased on pressure. A draught of cold milk, which happened to be at hand, was given to the woman, and produced relief. The loss of voice and the inability to swallow lasted three days; a large quantity of saliva with a bloody membrane was thrown off; the pain in the stomach continued. The patient recovered in a week. (*L'Union Médicale*, Fev. 19, 1857; *Brit. and For. Med. Rev.*, 1857, vol. 19, p. 500.)

A man, æt. 40, had been in the habit of taking one drachm of solution of ammonia diluted with water to relieve his breathing. In June, 1857, he took into his mouth by accident, an ounce of hartshorn spirit. He suddenly called for water and quickly ejected the fluid. He was immediately seized with intense burning pain and a feeling of suffocation. He thought that none of the fluid had reached the stomach. No antidote was administered. In two hours he was seen laboring under the following symptoms: countenance suffused, lips livid, breathing stridulous, aspect anxious, legs cold, beating the bed-clothes with his hands (malleatio!), pulse 100, the inside of the mouth, tongue, and throat, so far as could be seen, red, raw, and fiery-looking. There was pain referred to the larynx, but no pain in the stomach, even on pressure; there was no thirst. The symptoms of injury to the respiratory organs were the most marked. Under these, he sank on the twentieth day from laryngismus stridulus. (*Ed. Med. Jour.*, Sept. 1857, p. 236. Case by Dr. Paterson.)

Dr. Kern relates the case of a man, æt. 70, who took two mouthfuls of spirits of ammonia. He was immediately afterwards seized with a sense of suffocation, cough and vomiting, and in spite of treatment he died in four hours. The lining membrane of the mouth and throat was destroyed. There was a bloody fluid, smelling of ammonia, in the stomach. At the lower portion, the lining membrane was corroded, and the muscular coat changed into a black pulpy substance. The duodenum was also inflamed. (*Amer. Jour. Med. Sci.*, January, 1870, p. 275.) A man swallowed by mistake for a dose of cod-liver oil, a tablespoonful of solution of ammonia. Œdema of the glottis followed, and in five hours he died from suffocation. (*Lancet*, 1870, vol. 1, p. 467.) Eleven deaths from ammonia are reported to have occurred in England and Wales in four years—1863–7.

The following case occurred in Nov. 1858. A lunatic, æt. 62, swallowed about two fluid ounces of compound camphor liniment. The patient immediately complained of great heat in the stomach; vomiting was induced by giving to him warm water. The uvula, throat, and gullet were so intensely inflamed that he lost all power of swallowing; and the efforts to swallow liquids produced violent retching. The symptoms gradually abated, and the man recovered in four days. In this case the quantity of ammonia swallowed was small, amounting to about two and a half drachms, diluted with about six times the quantity of rectified spirit.

One of my pupils, Mr. Gill, communicated to me a case of the poisoning of an infant, only four and a half days old, by a small quantity of this liniment. The case occurred in September, 1863. Mr. Gill saw the infant about half an hour after the liquid had been taken; it was then screaming in a suppressed manner, as if the act increased the pain; the hands were tightly clenched; the skin was pale and covered with a cold perspiration; the mucous membrane of the lips was blistered, and that of the mouth and tongue was white. A yellowish froth escaped from the mouth and nostrils; breathing was painful, and the pulse imperceptible. In about two hours the infant appeared better, but at intervals it suddenly started and screamed, as if from sudden pain. In six hours it continued much in the same state, and swallowing was painful. In seventeen hours the skin was moist and cool; it had had a natural motion, and had been in a drowsy state during the night. After twenty-four hours the infant was much weaker; the limbs were cold, and the breathing was feebly performed. It became drowsy, and died thirty-two hours after taking the poison. There was an inquest, but no inspection of the body. A question of importance arose in reference to the case: namely, whether the mother or a child, two years of age, criminally administered the poison to the deceased infant. The mother stated that this child was playing with the bottle of embrocation on the bed, on which her infant was lying. She left the room for a short time, and on her return she gave the infant a teaspoonful of food which she had previously prepared for it. She was sure the infant swallowed part of the food; but as soon as the food was taken, it screamed violently and struggled for its breath, and then she perceived the food to smell strongly of the embrocation. As from the nature of this irritant compound the symptoms could not be suspended, it is clear that the mother either consciously or unconsciously gave the poison to her infant. On the latter supposition, it must have been placed in the food, which was on a chair near to the bed, by the child of two years, during her absence; but in this case it is remarkable that she did not perceive the odor until after she had poured the liquid into the mouth of the infant. The quantity swallowed was unknown. In the *Medical Times and Gazette*, for May 26, 1855, there are two cases reported, in which children were poisoned by swallowing a liniment of ammonia and oil. In one, an infant, death occurred speedily, probably from swelling and closure of the air-passages, thus leading to suffocation. In the other case, death took place on the following morning. Considering the hot taste of ammonia, it is remarkable that an infant could have had the power of swallowing nearly two ounces of strong ammoniacal liniment. It had been poured down its throat by another child of five years of age.

Serious injury to the organs of respiration is commonly the result of the local action of this poison, as in the following case, which was referred to me for examination by my colleague, Mr. Hilton, in May, 1857: A gentleman liable to attacks of fainting died in three days, after swallowing a quantity of a liquid administered to him by his son. This liquid, which was at the time believed to be sal volatile, was, in fact, a strong solution of ammonia. The deceased complained immediately of a sensation of choking and strangling in the act of vomiting. Symptoms of difficulty of breathing set in, with other signs of irritation in the throat and stomach. The mucous membrane of the mouth and throat was corroded and dissolved; and it was evident that the liquid had caused great local irritation. The difficulty of breathing was such as to threaten suffocation, and at one time it was thought that an operation must be resorted to. The state of the patient, however, precluded its performance, and he died on the third day. On inspection, the viscera presented strong marks of corrosion The covering of the tongue was softened and had peeled off; the lining membrane of the trachea and bronchi was softened and covered with layers of false membrane—the result of inflammation—the larger bronchial tubes were completely obstructed by casts or cylinders of this membrane. The lining membrane of the gullet was softened, and at the lower part, near its junction with the stomach, the tube was completely dissolved and destroyed. There was an aperture in the stomach in its anterior wall, about one inch and a half in diameter; the edges were soft, ragged, and blackened, presenting an appearance of solution. The contents of the stomach had escaped. On the inside, the vessels were injected with dark-colored blood, and there were numerous small effusions of red blood in various parts of the mucuous membrane. The coats were thinned and softened at the seat of the aperture. The blackened and congested appearances somewhat resembled that which is seen in poisoning by sulphuric or oxalic acid. The mucous matter on the coats of the stomach was feebly acid. No poison of any kind was found in the layer of mucus, or in the coats. There was not in any part the slightest trace of ammonia—the poison which had caused the mischief. The deceased had lived three days; remedies had been used, and every trace of ammonia had disappeared. The immediate cause of death was an obstruction of the air-tubes, as a result of inflammation, caused by the irritant action of the poison. It was quite obvious that a quantity of the liquid had entered the windpipe. The perforation of the stomach had probably taken place shortly before death, or there would have been marks of peritonitis. The injury to the stomach and œsophagus would have been sufficient to cause death, even supposing that the liquid had not penetrated into the lungs.

Solution of ammonia applied to the skin acts as a corrosive, and may inflame or cause the destruction of the parts which it touches. At the Stafford Summer Assizes, 1873 (*Reg.* v. *Gavan*), a man was convicted of throwing an ammoniacal liquid over the prosecutrix with intent to injure her. It was a liniment containing a strong solution of ammonia. The liquid was thrown in her face, and some portion reached the eyes; but she recovered from the effects. A weak solution acts as an irritant to the skin, while a strong solution causes vesication and a destruction of the part.

Fatal Dose.—The effects will depend more on the amount of injury to the air-passages and the stomach, than on the precise quantity taken. Death may be a very rapid result of the action of this poison.

The treatment in these cases must vary with the symptoms. Acetic acid and water, or lemon-juice with milk, or barley-water, may be given if the power of swallowing should exist.

CARBONATE OF AMMONIA—HARTSHORN—SAL VOLATILE.

Symptoms and Appearances.—The solution of this salt (sal volatile) is probably more active as a poison than is commonly supposed. The following case occurred in 1832: A man in a fit of passion swallowed about five fluid drachms of a solution of sal volatile. In ten minutes he was seized with stupor and insensibility; but, upon the application of stimulant remedies, he recovered. He suffered for some time afterwards from severe irritation about the throat and gullet. Dr. Iliff met with the case of a boy, æt. 2 years, who swallowed about half an ounce of spirit of hartshorn. He immediately screamed, and was very sick, bringing up some stringy mucus, at first of a light, and afterwards of a dark color. The lips were swollen; the breathing hard, hurried, and somewhat obstructed. There was perfect sensibility. The forepart of the mouth was but little affected; there was pain and difficulty in swallowing. The most urgent symptoms were connected with the lungs and air-passages. In three days the boy recovered. (*Lancet*, Dec. 1, 1849, p. 575.)

In a paper above referred to, Dr. Barclay relates the case of a girl, æt. 19, who, while in a state of unconsciousness, was made to swallow a quantity of hartshorn. She felt a severe pain in the stomach immediately afterwards, and in about an hour she vomited some blood. This vomiting of blood continued for several days. These symptoms were followed by great irritability of the stomach, and the constant rejection of food. There was obstinate constipation of the bowels, with great emaciation and loss of strength. She died in about three months from the time at which she had swallowed the alkaline poison. On inspection, the gullet was found healthy; the orifice, at its junction with the stomach, was slightly contracted. The pyloric opening was reduced to the size of a crowquill, and the coats were thickened. On the posterior wall of the stomach there was a dense cicatrix of the size of half a crown, and from this point, fibrous bands ramified in various directions. The duodenum and other parts of the intestinal canal were healthy. (*Med. Times and Gazette*, Nov. 26, 1853, p. 554.) A case occurred to Mr. Procter, in May, 1852, in which a woman gave to her infant, 4 weeks old, a teaspoonful of hartshorn of the strength of about nine per cent. The child became more and more depressed, and died thirty-six hours after taking the liquid. There was no vomiting or purging, and the mouth and throat presented no excoriation; there was, however, slightly increased redness of the lining membrane. An examination after death was not made.

The salts of ammonia are not often used by persons who are intent upon suicide or murder, but there is one instance on record in which a man was tried for the murder of a child by administering to it spirits of hartshorn. (*Regina* v. *Haydon*, Somerset Spring Assizes, 1845.) Of the action of the other compounds of ammonia on man, nothing is known.

Chemical Analysis.—The three alkalies, potash, soda, and ammonia, are known from the solutions of the alkaline earths, by the fact that they are not precipitated by a solution of carbonate of potash. They all three possess a powerful alkaline reaction on test-paper, which, in the case of ammonia, is easily dissipated by heat. *Ammonia* is immediately known from potash and soda by its odor and volatility. If the solution in water be very dilute, the odor may be scarcely perceptible. The alkali may then be discovered, provided we have first assured ourselves, by evaporating to dryness a portion of the liquid, that other alkalies and alkaline salts are absent, by adding to the solution a mixture of arsenious acid and nitrate of silver, when the well-known yellow precipitate of arsenite of silver will be instantly produced. The same result takes place when a carbonate (even bicarbonate of lime) is present; but if a carbonate or other salt existed in the liquid, it would be left on evaporation. In addition to these characters, ammonia redissolves the brown oxide of silver, which it precipitates from the nitrate, while potash and soda do not. (For the detection of ammonia in *organic mixtures*, see *ante*, p. 251.)

The *carbonate of ammonia* may be known from other salts by its alkaline reaction, its odor, and its entire volatility as a solid: from pure ammonia—1. By its solution effervescing on being added to an acid; 2. By its yielding an abundant white precipitate with a solution of chloride of calcium; from the carbonates of potash and soda, among other properties—1. By its giving no precipitate with a solution of the sulphate of magnesia; 2. By the rich violet blue solution which it forms when added in excess to a weak solution of sulphate of copper; 3. By its odor and volatility.

[*Spirit of hartshorn* is a name applied either to ammonia, carbonate of ammonia, or a mixture of the two.]

CHAPTER XXX.

POISONING BY SALINE SUBSTANCES—ACID OXALATE OF POTASH (SALT OF SORREL)—ACID TARTRATE OF POTASH (CREAM OF TARTAR)—NITRATE OF POTASH—SULPHATE OF POTASH—SULPHATE OF ALUMINA AND POTASH (ALUM)—IODIDE OF POTASSIUM—THE SALTS OF BARIUM.

SOME saline compounds have been found to exert an irritant action on the system. The pure alkalies and their carbonates have a corrosive (chemical) action when concentrated, but they operate as irritants when diluted. The salts about to be described are not very energetic poisons, and, with one or two exceptions, require to be given in large doses in order to produce noxious effects.

ACID OXALATE OF POTASH, OR SALT OF SORREL.

The poisonous effects of this salt depend entirely on the oxalic acid which it contains. It is much used for the purpose of bleaching straw and removing ink-stains, being sometimes sold under the name of essential salt of lemons. The smallest quantity retailed to the public, is a quarter of an ounce, and for this three halfpence is charged. Its poisonous properties are not commonly known, or no doubt it would be frequently substituted for oxalic acid. There are now a sufficient number of cases on record to show that this salt is a powerful poison.

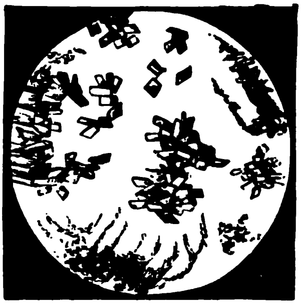
Symptoms and Appearances.—A young lady, æt. 20, swallowed an ounce of the acid oxalate dissolved in warm water. She was not seen by any one for an hour and a half; she was then found on the floor, faint and exhausted, having previously vomited considerably. There was great depression, the skin cold and clammy, the pulse feeble, and there was a scalding sensation in the throat and stomach. There was also continued shivering. Proper medical treatment was adopted, and she recovered in two days, still suffering from debility and great irritation of the stomach. During the state of depression, it was remarked that the conjunctivæ were much reddened, and the pupils dilated. There was great dimness of vision. (*Med. Gaz.*, vol. 27, p. 480.) In another case, two hundred and twenty-five grains were taken (about half an ounce). Bicarbonate of soda was given as an antidote, and the patient completely recovered. (*Med. Times and Gaz.*, Feb. 12, 1859.) The recovery must have taken place in spite of the antidote, for the oxalate of soda is just as poisonous as the oxalate of potash. For a third case of recovery, see the same journal, Oct. 15, 1859, p. 378. A woman swallowed about a quarter of an ounce of "salts of lemon." In two or three minutes she threw up her arms and fell down in a state of insensibility. An emetic was given, and in about an hour she partially recovered her consciousness. She was admitted into Guy's Hospital an hour and a half after taking the poison. She was then partly conscious. Chalk mixture was given to her, and the stomach-pump was used, as there had been no vomiting. In two days she had recovered. (*Guy's Hosp. Rep.*, 1874, p. 416.)

Among the fatal cases may be mentioned the following: A lady, recently confined, took by mistake half an ounce of the acid oxalate, instead of cream of tartar. She had scarcely swallowed the draught, when she was seized with violent pain in the abdomen and convulsions; she died in *eight minutes*. On inspection the mucous membrane of the stomach and small intestines was found inflamed. (*Ann. d'Hyg.*, Avril, 1842.) A case is reported by M. Chevallier, in which death took place in *ten minutes*. (*Ann. d'Hyg.*, 1850, vol. 1, p. 162.) In one instance, in which it was supplied by mistake for Epsom salts, it caused death in an hour and a half (*Pharm. Jour.*, March, 1872, p. 760); and in another, a teaspoonful of the salt taken for three successive mornings, produced severe vomiting. In about an hour after the third dose the patient expired. We learn from these cases that this salt is poisonous, destroying life almost as rapidly as oxalic acid itself; and that in the symptoms which it produces, it closely resembles this poison. In the shortest fatal case half an ounce killed an adult in *eight minutes;* but probably the fatal effects were in this instance accelerated by the debilitated state of the person who took it.

In a case reported in the *Ed. Monthly Journal*, July, 1862, p. 92, death appears to have been caused by this salt as the result of *chronic* poisoning. A girl was charged with the murder of her father. He began to be ill about December 5, and he died on January 26 following. He suffered from vomiting, heat, and irritation in the mouth and throat, prostration of strength and constant pains in the chest and abdomen. After death, the appearances were: Inflammation of the mucous membrane of the stomach and part of the bowels. They contained a dark-colored fluid. The mucous membrane of the gullet was destroyed. The coats of the stomach, which were thickened and injected, had a gangrenous appearance. There was no proof that the prisoner had had possession of the poison until January 11, five weeks after the symptoms had begun in the deceased. The symptoms before and subsequently to this date were similar. This absence of proof of possession, led to the acquittal of the prisoner; still it would be difficult to account for the symptoms and appearances on any theory of disease.

Chemical Analysis.—This salt is not very soluble in cold water, but its solution may be readily mistaken for that of oxalic acid. It is not dissolved by alcohol. 1st, the aqueous solution has an acid reaction; and, 2d, it is precipitated by nitrate of silver and sulphate of lime, like oxalic acid; but with the latter test the precipitation is much more copious. It is distinguished from oxalic acid: 1. By its crystals, which when slowly produced on a glass slide, assume the shape of small rhombic prisms, sometimes grouped in a plumose form; and 2. By heating a portion on platinum-foil. While oxalic acid is entirely volatile, the acid oxalate leaves an ash, which, when sufficiently heated, is white and alkaline. This may be proved to contain carbonate of potash by its dissolving with effervescence in diluted nitric acid, and forming nitrate of potash.

FIG. 12.



Crystals of acid oxalate of potash, magnified 30 diameters.

In some instances this poisonous salt has been supplied by mistake for cream of tartar, and has caused death. Cream of tartar, or acid tartrate of potash, leaves a black alkaline residue when heated in close vessels. Its solution is less acid than that of the salt of sorrel. It is not precipitated by the nitrate of silver or sulphate of lime. The different action of the two salts on writing ink affords a simple means of identification. The acid oxalate of potash immediately discharges the color of ink when warmed, while the acid tartrate does not possess this property.

In organic mixtures the process is the same as for oxalic acid itself, but, owing to its insolubility, a portion of the salt may be found as a sediment in the fluids of the stomach or in the liquid vomited. Although the salt is a natural constituent of sorrel, this vegetable is rarely used as an article of food in England, and then only in small quantity. According to Mitscherlich, the proportion of acid oxalate is only 0.75 per cent. of the weight of the fresh plant, or 3.75 per cent. of the juice; hence, one ounce of fresh sorrel will yield but little more than three grains of the salt. The objection suggested by Orfila, that the salt found in a dead stomach, might be due to the presence of sorrel taken in soup, is therefore inadmissible, except when found only in traces, and no symptoms of poisoning have existed.

ACID TARTRATE OF POTASH—CREAM OF TARTAR—ARGOL.

Symptoms and Appearances.—This salt, although not commonly regarded as a poison, has proved fatal in at least one instance. The case occurred in this metropolis in 1837. A man, aged 37, took four or five tablespoonfuls of cream of tartar. He was seized with violent vomiting and purging. There was pain in the abdomen, thirst, feeble pulse, and the thighs and legs appeared paralyzed. The fluid vomited was of a dark-green color, and the motions had the color of coffee-grounds. Death took place in about forty-eight hours. On inspection, the mucous membrane of the stomach and duodenum was found highly inflamed, the cardiac portion of the former being of a deep red color, with some spots of black extravasation. The stomach contained a thick brown fluid, colored by bile. The whole of the intestinal canal was more or less inflamed.

According to Wöhler, this salt passes off by the urine, under the form of carbonate of potash, the secretion being alkaline. Belloc relates a case of alleged poisoning by Rochelle salts, the compound tartrate of potash and soda. The circumstances, however, render his statement somewhat doubtful. (*Cours de Méd. Leg.*, 139.)

Chemical Analysis.—The chemical properties of this salt have been given above, p. 260. *Organic mixtures.*—As it is comparatively insoluble in water, it may be found as a sediment at the bottom of a liquid. If dissolved, the liquid may be concentrated and alcohol added; cream of tartar is insoluble in alcohol, and organic matter may be thus separated from it. If the organic liquid is strongly colored, purified animal charcoal may be used to decolorize it, or the liquid may be dialyzed (p. 154). In detecting this substance in the stomach, it is proper to bear in mind that it is a natural constituent of the potato.

NITRATE OF POTASH—NITRE—SALTPETRE—SAL PRUNELLA.

This well-known salt is largely employed in the arts. It is an irritant, but acts only as such when taken in a large dose. It has destroyed life on several occasions. Its effects are, however, somewhat uncertain. An ounce, and even two ounces, have been taken without causing very alarming symptoms. (*Ed. M. and S. J.*, vol. 14, p. 34.) Dr. Bennett states that M. Gendrin was in the habit of giving it in doses varying from six to twelve, or sixteen drachms in the twenty-four hours without any dangerous symptoms resulting. (*Med.-Chir. Review*, April, 1844, p. 549.) M. Mozade has given it with benefit in cases of dropsy in from three to five drachm doses. (*L'Union Médicale*, 3 Juin, 1847, p. 274.) If the doses are not excessive or repeated at too short intervals, a large quantity of nitre may be thus safely passed through the body, and produce a beneficial operation. In half-drachm, drachm, and drachm and a half doses, taken thrice daily, Dr. Wilks gave to a man, æt. 34, nearly twenty-six ounces of nitre over a period of forty-six days. It was freely eliminated in the urine, and did no injury to the patient. (*Guy's Hosp. Rep.*, 1863, p. 173.) According to Tourtelle, no injury has followed even in cases where it was given in doses of an ounce. (Galtier, *Toxicologie*, vol. 1, p. 268.) Tartra denied that it had poisonous properties even in a very large dose (*Op. cit.*, p. 135); but cases have occurred which now leave no doubt upon the subject. Three deaths from this salt are reported to have taken place in four years, 1863–7.

Symptoms and Appearances.—In one instance, quoted by Orfila, an ounce of nitre was taken by a lady in mistake for other salts. In a quarter of an hour, she suffered from nausea, vomiting, and purging; and the muscles of the face were convulsed. The pulse was weak, the respiration laborious, the limbs cold, and there was a sense of burning heat and severe pain at the pit of the stomach. She died in *three hours* after taking the dose. On inspection, the stomach was found highly inflamed, and the membrane detached in various parts. Near the pylorus, the inflammation had a gangrenous character. A large quantity of liquid mixed with blood was found in the stomach. (Vol. 1, p. 283.) In another case, which proved fatal in sixty hours, where an ounce and a half of nitre had been taken, a small perforation was found in the stomach. (*Ibid.*) I am indebted to the late Dr. Geoghegan, of Dublin, for the following case: A man took from an ounce to an ounce and a half of nitre by mistake for salts. Severe pain in the abdomen followed, with violent vomiting, but no purging as far as could be ascertained. He died about *two hours* after taking the salt. On examining the body, a bloody mucus was found in the stomach; the lining membrane was of a brownish-red color, generally inflamed, and in parts detached from the coat beneath. None of the poison could be detected in the stomach; but its nature was clearly established from the analysis of a portion left in the vessel which had contained the draught. Two men swallowed, each, once ounce of nitre by mistake for Glauber's salt. They almost immedietely experienced a sense of coldness in the course of the spine, trembling in the limbs, with violent vomiting and purging. The evacuations were bloody. They recovered in the course of a few days. (Casper's *Wochenschrift*, No. 18, 1841.) A case is reported in the same journal, in which one ounce of nitre killed a man in thirty-six hours. In another case an old man, æt. 60, lost his life from an overdose of nitre which he had taken as a medicine. The dose amounted to about ten drachms; it caused profuse purging and death in about five hours. Death was referred to inflammation of the mucous membrane of the stomach and bowels, owing to the irritant action of the nitre.

A woman, æt. 28, swallowed in two doses, taken on two days, about an ounce of nitrate of potash. After the second dose, she was attacked with severe burning pain in the stomach, and with violent vomiting, followed by collapse. There was no purging, and the secretion of urine was arrested, although the salt is generally regarded and employed as a diuretic. The woman recovered in a few days. (*Pharm. Jour.*, Feb. 1846, p. 356.) Mr. Gillard met with a case in which a man recovered in four days after having swallowed two ounces of nitrate of potash by mistake for Epsom salts. In about five minutes after taking the nitre, he felt a burning pain in his stomach, and this was immediately followed by sickness. Free vomiting was excited by mustard; this probably led to his recovery. (*Prov. Med. Journ.*, Aug. 19, 1846, p. 382.)

Other cases of recovery after large doses are reported. A man, æt. 30, who had taken nitre medicinally—half an ounce in divided doses in the twenty-four hours—took twelve doses at once. He immediately became insensible, and his face was pale and collapsed as in cholera. The skin was of a marble coldness, the pulse slow, small, and feeble, as was also the respiration. An ipecacuanha emetic restored the power of speech; but he complained of severe burning pains in the throat and abdomen, and blood was passed in the evacuations and urine. There was trembling, with slight convulsions, which, together with hallucinations of the senses, and a partial paralysis of the muscles, indicated an operation of the nitre on the nervous system. Free local bleeding, anodyne poultices, and abundant drinks and enemata of milk and linseed-tea, were directed against the inflammatory action, while the great depression and other nervous symptoms were met with camphor and ether. Although dangerous symptoms were thus dissipated, the patient long suffered from derangement of the digestive and urinary organs, and complained of a peculiar feeling of coldness in his hands and feet and down the back. (*Berlin Med. Zeitung*, 1855, No. 49; *Med. Times and Gazette*, Aug. 30, 1856.) A woman swallowed an ounce of nitre in two teacupfuls of water. She immediately vomited. When seen the following day, she was sitting with her legs drawn up, the surface of the body pale, but warm and moist. There was constant vomiting. The abdomen was swollen, but soft and tender to the touch. She was unable to move for a week, and then suffered from severe purging with griping; the evacuations were bloody. She recovered, but remained weak for a long period. (*Med. Times and Gazette*, Nov. 7, 1857, p. 484.)

Mr. Fuller, of Oswestry, communicated to me a case which proved fatal in December, 1863. A man swallowed an ounce of nitre, mixed with water, by mistake for Epsom salts, about nine o'clock in the morning. It produced vomiting with severe pain, but no purging. There was coldness of the surface and lividity of the face. Death took place in three hours. On inspection the mucous membrane of the stomach was found highly inflamed, especially towards the middle of the greater curvature, where for several inches it resembled scarlet cloth. The pylorus and duodenum were of a deep crimson color. The peritoneal surface was very vascular, especially over the stomach, the vessels having a vermilion red color, as if they had been injected. The heart and lungs were healthy, the blood was fluid and more florid than natural. The other organs presented no unusual appearance. No analysis was made of the contents of the stomach, but that the nitre was the cause of death no doubt could be entertained, and a verdict was returned accordingly at the coroner's inquest. These facts show that the effects of nitre, although serious, are very uncertain in their character and duration.

Poisoning by nitre has been hitherto the result of accident. It is not taken for the purpose of suicide, the popular opinion being that it is not poisonous; although the above cases show that in a large dose it may destroy life with greater rapidity than arsenic or corrosive sublimate. It is not likely to be employed by a murderer, since a dose sufficient to kill, could not be administered in any article of food without giving warning by the taste.

Treatment.—There is no antidote known. Mucilaginous drinks should be given; vomiting should be freely promoted, and the stomach-pump used for the removal of the salt.

Chemical Analysis.—(See NITRIC ACID, *ante*, p. 210.) The process of dialysis (p. 154) may be employed for the separation of the salt from the contents of the stomach, when mixed with liquid food, blood, or mucus. Prismatic crystals of nitre are readily obtained by evaporating the dialyzed liquid. (See Fig. 6, p. 211.) There is no doubt that nitre is eliminated largely in the urine. A case is elsewhere reported in which nitre was separated from the urine of a person who had been taking it for many weeks medicinally. (See *ante*, p. 39.) Orfila states that he detected nitre in the liver, spleen, kidneys, and urine of animals poisoned by it. (*Ann. d'Hyg.*, 1842, vol. 2, p. 434.)

SULPHATE OF POTASH—SAL POLYCHREST—SAL DE DUOBUS.

This salt was, at one time, regarded as inert, but of late years the employment of it in medicine has given rise to some important medico-legal investigations.

Symptoms and Appearances.—A lady, about a week after her delivery, took, by the prescription of her medical attendant, about ten drachms of the sulphate of potash in divided doses, as a laxative. After the first dose, she was seized with severe pain in the stomach, nausea, vomiting, purging, and cramps in the limbs. These symptoms were aggravated after each dose, and she died in *two hours*. It was supposed that some poison had been given by mistake; but there was no evidence of this, and the question really was, whether her death had or had not been caused by the sulphate of potash. On an inspection of the body, the mucous membrane of the stomach and intestines was found pale, except the valvulæ conniventes (folds), which were reddened. The stomach contained a large quantity of reddish-colored liquid, which, on analysis, was found to contain only sulphate of potash, and no trace of any common irritant poison. The examiners referred death to sulphate of potash taken in an unusually large dose, whereby it had acted as an irritant poison on a person whose constitution was already much debilitated. (*Ann. d'Hyg.*, Avril, 1842.)

The question whether this is to be regarded as an irritant poisonous salt or not, was much debated among members of the profession, in reference to a case which was tried at the Central Criminal Court in October, 1843. (*The Queen* v. *Haynes.*) The accused had given to the deceased, on the night before her death, two ounces of sulphate of potash, dissolved in water; and it was alleged that a fortnight previously to this, she had taken in divided doses, as much as a quarter of a pound of this salt. The woman thought that she was pregnant, but this was disproved by an examination of the body; and it was charged that the prisoner had given her the salt with the intention of causing a miscarriage. After the last dose, she was seized with sickness, and died within a very short time. The stomach was found empty, but highly inflamed; and there was blood effused on the brain. One medical witness referred death to the action of the sulphate as an irritant poison; the other to apoplexy, as an indirect result to the violent vomiting caused by it. The prisoner was acquitted of the charge of murder, but subsequently found guilty of administering the sulphate with intent to procure abortion. Both of the witnesses admitted that, in small doses, the salt was innoxious; but that in a dose of two ounces it would produce dangerous effects. A portion of the sulphate in this case was examined by the late Mr. Brande, as it was suspected that some poisonous substances might have become accidentally mixed with it; but it was found to be pure. It is not improbable, from the symptoms and the inflamed state of the stomach, that the salt acted here as an irritant poison; and the fact of its being a proper medicine in small doses, appears to be no sound objection to this view; for the same circumstances is observed with respect to numerous substances, the poisonous or noxious properties of which cannot admit of dispute.

A case, somewhat similar in its details, was the subject of a trial at the Central Criminal Court in October, 1856. (*Reg.* v. *Gaylor.*) A married woman, the wife of the prisoner, under the belief that she was pregnant, took a large quantity of this salt, the prisoner having purchased two ounces, and handed it to her. It was taken with the design of procuring abortion, but it caused the death of the woman under symptoms of severe irritation of the stomach and bowels. The deceased was not seen by a medical man while living, but she suffered from severe pain, vomiting, and purging; the vomited matter had a bilious color. On inspection, the stomach and the upper portion of the small intestines were of a deep purple color, as if from the action of some irritant substance. The stomach, when opened, showed marks of irritation, and its mucous coat was much congested. In this organ there was a spoonful of thick, slimy fluid, in which a quantity of sulphate of potash was found. The intestines contained twelve ounces of a thick white fluid, highly charged with mucus, and this, when analyzed, yielded sulphate of potash.

There was no doubt that death had been caused by an overdose of this substance; but a legal doubt was raised whether the prisoner had committed any crime in handing it to the deceased. According to Mr. Mowbray (*Medical Gazette*, vol. 33, p. 54), sulphate of potash is a salt much employed in France as a popular abortive. He quotes several instances in which, in large doses, it produced severe symptoms, resembling those of irritant poisoning, and even death. In one case, two drachms acted powerfully; and in another, that fell under his own observation, four drachms of the salt, administered to a lady after her confinement, had all the effects of an irritant poison. The above cases are the only instances in which, I believe, it is publicly known to have proved fatal in England; and they show that substances, commonly reputed as innoxious, may give rise to important questions in toxicology. There is no doubt that the most simple purgative salts may, under certain circumstances, and when given in large doses, destroy life. A case is reported in which sulphate of magnesia caused death, and gave rise to a criminal charge in this country.

It is said that sulphate of potash has, in some cases, caused vomiting and other serious symptoms, from its containing as impurity sulphate of zinc. This, if present, would be easily discovered by the appropriate tests. A more dangerous impurity has been lately detected in it by M. Bussy, namely, the arseniate of potash. He found this poison in a sample of sulphate, supplied by a wholesale house in Paris. (*Pharm. Jour.*, May, 1872, p. 954; also *Ann. d'Hyg.*, 1872, vol. 2, p. 137.) It may have been derived from arsenical sulphuric acid used in its manufacture. It would be proper to test for arsenic any sample of sulphate which has caused great irritation. (See ARSENIC.) Arsenic may thus find its way into all medicines in which sulphate of potash is used, *e. g.*, the compound colocynth pill and the compound powder of ipecacuanha.

Chemical Analysis.—Sulphate of potash is easily identified. It is a dry hard salt, soluble in water, forming a neutral solution. This solution, if sufficiently concentrated, is precipitated both by tartaric acid and chloride of platinum, whereby potash is indicated (p. 250); and the presence of sulphuric acid is known by the action of a salt of barium (p. 196). *Organic liquids.*—This salt being insoluble in alcohol, may have the organic matter removed from it by treating the liquid containing it (previously concentrated) with alcohol, or the substance containing the salt may be evaporated to dryness and incinerated, when the undecomposed sulphate may be obtained by lixiviating the calcined residue with distilled water. The sulphate of potash exists naturally in some animal fluids, but only in traces. It may be separated from organic substances by dialysis (*ante*, p. 154).

ALUM—SULPHATE OF ALUMINA AND POTASH.

This substance is very commonly diffused, but it does not appear to have given rise to any accidents in this country. One case of death from alum appeared in the Registration Returns for 1838–9. A singular case occurred in Paris, in 1828, in which the alleged noxious properties of alum were brought into question. A lady swallowed a quantity of calcined alum dissolved in warm water, which had been supplied to her by mistake for powdered gum. The quantity taken was less than half an ounce. She immediately complained of a burning pain in the mouth, throat, and stomach. She afterwards suffered from thirst, violent vomiting, and general disturbance of the system, from which she recovered in the course of two or three days. These effects were referred to the alum, and the person who supplied it by mistake was condemned to a severe punishment. On the case being carried to an appeal, Orfila contended that alum was not a poison; although he admitted that in the calcined state it was used as a caustic. In order to establish his opinion of its inertness, he offered to swallow half an ounce on the spot! He referred the symptoms under which the patient had labored to some other cause; but on being further questioned, he admitted that a solution of calcined alum was likely to produce more serious effects than common gum, which the party should have taken. The punishment was mitigated. (*Ann. d'Hyg.*, 1822, vol. 1, p. 234.) Orfila subsequently ascertained by experiment that alum in a large dose operated fatally on animals, destroying life in the course of a few hours! He detected the salt in these cases in the substance of the stomach, liver, spleen, and in the urine. (*Ann. d'Hyg.*, 1845, vol. 2, p. 433.) The reader will find a singular case of supposed poisoning by alum in the *Ann. d'Hyg.*, 1832, vol. 2, p. 180.

The *symptoms* produced by alum in a large dose are frothing at the mouth, vomiting (the vomited matters containing alum), purging, depression, weakness of the limbs, and the principal *appearance* is a reddish-brown color of the mucous membrane of the stomach, which may be found softened or disorganized, either wholly or in patches. We cannot therefore refuse to admit the fact of this substance acting as an irritant. It is, however, proper to observe that alum, given in large doses to animals, does not appear to affect them seriously, unless the gullet has been tied; *three drachms*, dissolved in six ounces of liquid, have been given at a dose without any inconvenience resulting.

Treatment.—The promotion of vomiting and the free administration of hydrate of magnesia, or a weak solution of carbonate of ammonia at intervals, with the use of the stomach-pump.

Chemical Analysis.—Common alum possesses a peculiarly acid and astringent taste. It is easily dissolved by water, forming an acid solution, which crystallizes on evaporation in regular octahedra. Its solution is not affected by ferrocyanide of potassium or sulphuretted hydrogen gas, whereby it is known from the true metallic saline solutions. The sulphuric acid may be detected in the solution by a salt of barium. On adding potash, a white precipitate of alumina falls down, which is redissolved by the addition of a larger quantity of the alkali. By this last character, it is known from the alkaline earths, which are precipitated from their solutions by potash, but the precipitates are not redissolved. On adding carbonate of ammonia, alumina falls down. This may be separated by filtration, and on evaporating the liquid portion, and incinerating the saline residue, there will be found sulphate of potash. *Calcined* alum is a white uncrystalline substance. It is used as a mild escharotic, and is only partially soluble in water. About one-sixth is left as a residuary white powder, easily soluble in a mineral acid, and yielding common alum by crystallization. The quantity dissolved by boiling water is, however, sufficient to allow its nature to be determined. From *organic liquids* it may be obtained by evaporation and incineration.

IODIDE OF POTASSIUM.

Iodism—Symptoms.—This salt is extensively employed as a medicinal preparation, but it appears to have given rise, in some instances, to alarming symptoms, even when exhibited in small doses; and it is stated that death has resulted from its use. The following cases may serve to illustrate its alleged noxious effects. A gentleman was ordered by his physician to take three grains of the iodide in a draught of peppermint-water three times a day. After the third dose he felt unwell, and an hour after the fourth dose he was attacked with a violent shivering fit, followed by headache, hot skin, intense thirst, quick and full pulse, with vomiting and purging. These symptoms were succeeded by great prostration of strength. In spite of treatment, the purging lasted several days. The effects of the medicine in this case were so violent, although only *twelve grains* had been taken, that there is little doubt, if the patient had taken another dose, he would have died. (*Med. Gaz.*, Sept. 3, 1841.) In October, 1841, a case was reported by Mr. Erichsen to the University College Medical Society, in which alarming symptoms resulted from a dose of only *five grains* of iodide of potassium. There was great difficulty of breathing, discharge from the eyes and nostrils, inflamed conjunctivæ, and most of the violent symptoms of catarrh. The iodide was discontinued, and the patient recovered. Dr. Lawrie found that seven grains and a half of the iodide, in three doses, produced in an adult, dryness and irritation of the throat, great difficulty in breathing, and other serious symptoms. In another instance, thirty grains, in divided doses, caused severe headache and secretion of tears. In two instances, wherein he had prescribed it medicinally in small doses, it was, in his opinion, the cause of death. (*Med. Gaz.*, vol. 27, p. 588.)

Mr. Cooper Forster describes in the *Guy's Hosp. Rep.* (1873, p. 41), the following case, in which forty grains taken in two doses produced serious effects.

A man, æt. 43, was under his care for chronic ulcers of the legs. On August 19, he was ordered one scruple of the iodide of potassium in one ounce of infusion of gentian three times a day. The first dose was taken on the morning of August 20, the second at 2 P.M. Within half an hour after the first dose he began to sneeze very much, and this symptom continued until the second dose, after taking which his eyes began to water, his head to ache severely, and his throat to become very sore.

The next day the eyelids were swollen so as to close the eyes, and there was a thick purulent discharge from the conjunctivæ. The skin of the nose was red, with purulent patches upon it, the mucous membrane discharging a watery fluid to a large extent. The throat was sore; the fauces were rather injected. The voice was only a hoarse whisper. There was much pain in all the limbs. On the 24th these symptoms had disappeared. The man took only 40 grains in two doses, and these were the effects. He at first presented the appearance of one suffering from erysipelas, but the skin had not the usual redness, and his temperature remained below the normal. One of the effects of this compound, according to Marotte, was the production of a great enlargement of the thyroid gland. (Bouchardat, *Ann. de Thérap.*, 1873, p. 221.)

These cases show the necessity of caution in the medicinal use of this substance. The effects from small doses may, perhaps, be attributed to idiosyncrasy; still there seems to be good ground, from the results of experiments on animals, for ranking iodide of potassium among noxious irritant substances. It has not, so far as I know, caused death, if we except the two cases recorded by Dr. Lawrie. One drachm and a half of the solution has been taken by a young woman without destroying life, although it produced serious symptoms of irritation. (Devergie, *Méd. Lég.*, vol. 2, p. 535.) It has been suggested that the occasional adulteration of the iodide with carbonate of potash may account for the discrepant statements respecting its innoxious properties in large doses. In one instance, in which the medicinal dose had been carried to several drachms, the iodide was found to contain 75 per cent. of the carbonate of potash. This may explain the fact that large doses of the iodide have been given by French surgeons in the treatment of syphilis without producing injurious consequences. M. Payen has prescribed as much as 60 grains daily in divided doses, and M. Ricord is stated to have carried the dose to 135 grains in a day. Another theory, however, may be adopted to account for the innoxious character of these large doses. A state of tolerance may have been set up as in the administration of tartar emetic in cases of pulmonary disease.

Chemical Analysis.—The iodide may be distinguished by its cubic crystals and by its solution producing a blue color with starch on the addition of strong nitric acid. The salt gives a violet color to flame, indicative of potash, and yields iodine when treated with sulphuric acid and oxide of manganese.

The detection of this salt when dissolved in organic liquids—in the contents of the stomach, in the urine, or the tissues, is based on very simple principles. We make a watery extract, or evaporate the liquid to an extract, and distil this with a solution of perchloride of iron. If the iodide is present, even in small quantity, the vapor of iodine is distilled over and is easily recognized by its color, odor, and reaction on starch-paper. The presence of organic matter does not interfere with this result, and an excess of the perchloride does not prevent the whole of the iodine being set free.

A preliminary experiment may be made with a portion of the liquid in a test-tube.

SALTS OF BARIUM—CHLORIDE, NITRATE, AND CARBONATE.

Symptoms and Appearances.—A woman, æt. 23, took by mistake for Epsom salts, less than a teaspoonful (100 grains) of the chloride of barium. This was at 12.30 P.M. In half an hour there was a feeling of deadly sickness, with sharp burning pains in the stomach and bowels. Vomiting and purging set in violently, the purging being attended with tenesmus. An hour and a half after she had taken the poison, the following symptoms were observed by Mr. Walsh. Face pale and anxious, eyes deeply sunk, surface cold, heart's action feeble, pulse scarcely perceptible, tongue natural and warm, loss of muscular power, sensation and intelligence not affected, pupils natural. Fluids taken were instantly rejected with a ropy mucus. There was pain in the stomach, a ringing in the ears, twitching of the face, and twisting of the legs and arms. At 9 P.M., the symptoms had abated, but at 2 A.M. (*i. e.*, in about fourteen hours) the purging had returned, and the symptoms were much worse. There was a loss of voluntary muscular power. The breathing was slow and labored, and indicated effusion in the bronchial tubes, but the woman was sensible. Soon after 3 A.M. she was convulsed, and these convulsions continued in paroxysms for two hours, when she died, seventeen hours after taking the poison. During the fits she had several watery evacuations, and consciousness was lost. There was no post-mortem examination. (*Lancet*, 1859, vol. 1, p. 211.) Giddiness, convulsions, and paralysis have been remarked among the symptoms. A recent instance of death from the chloride of barium is reported in the *Pharmaceutical Journal* (Aug. 10, 1872, p. 117); but no account is given of the dose taken, or of the symptoms and appearances. Mr. Kennedy states that in using this compound as a medicine, he has found that few persons are able to bear the eighth of a grain; that it is analogous to corrosive sublimate, and that an overdose will produce similar effects. He has used it for many years, and he finds the proper dose is from the twelfth to the sixteenth part of a grain; but he cites no instance of its acting as a poison in a dose of one or two grains. (*Lancet*, July 5, 1873, p. 28.)

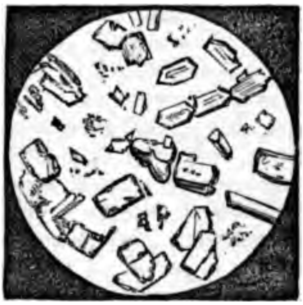
The following case is reported by Wildberg: A woman swallowed, by mistake, half an ounce of powdered chloride of barium dissolved in warm water. Nausea and vomiting of a watery mucus supervened, with twitchings of the facial muscles, and convulsive motions of the hands and feet. The symptoms continued to increase in severity, and she died in about *two hours* from the time of taking the poison, under the most violent convulsions. On inspection, the stomach was found perforated posteriorly, in the lesser curvature, near the cardiac orifice. The aperture was of an oval form, three lines in diameter externally, and almost twice as large internally. The margin of the aperture appeared swollen, and the mucous membrane for about two inches around was much thickened and covered with a bloody mucus. The stomach and small intestines were highly inflamed; the cavity of the former contained mucus and coagulated blood. The pharynx and œsophagus presented slight marks of inflammation. The poison was detected in the stomach by chemical analysis. Wildberg suggested that the perforation was due to disease, and not to the poison taken. This is very probable, for the characters of the aperture were those of perforation from disease (*ante*, p. 129); and it would be unlikely that the chloride of barium, if it led to perforation of the stomach at all, should have given rise to this effect in two hours. It is not stated whether the woman had suffered from any symptoms of gastric irritation prior to taking the poison, nor whether the contents of the stomach were found extravasated and the peritoneum inflamed. But there can be no doubt that she died from the effects of the poison. This was clearly indicated by the nature of the symptoms, and the appearances after death. Admitting that no mistake was made respecting the time at which it was swallowed, it must be considered remarkable that this substance should have destroyed life, and left such extensive marks of irritation in the alimentary canal, within the short space of two hours. In another instance, one ounce of this salt, taken by mistake for Glauber's salt, caused death in an hour. Even in small doses, the chloride of barium has been observed to affect the system powerfully. Orfila found that the chloride was absorbed; he detected it in the liver, spleen, and kidneys of animals poisoned by it. (*Ann. d'Hyg.*, 1842, vol. 2, p. 217.)

A fatal case of poisoning with *nitrate of baryta*, taken in mistake for sulphur, is reported in the *Pharmaceutical Journal* for 1869, p. 181. Another case, also fatal, is recorded in the same journal for June, 1872, p. 1021. It appears that the salts of barium are used for sizing cotton warps. A man employed in this work swallowed a portion about the size of a bean thinking he was taking Epsom salts. He died in about fourteen hours. The symptoms were at first those of irritant poisoning, but in the latter stage paralysis took place. The medical witness stated that he had found twelve grains of these salts sufficient to kill a

M. Chevallier met with a case in which *acetate of baryta* had upplied as a medicine in place of the sulphovinate of soda. It the death of the patient, and produced serious symptoms in the ist. He had swallowed a portion of the medicine, in order to hat there had been no mistake in its preparation. (*Ann. d'Hyg.*, vol. 1, p. 395.) An action was brought by the widow against uggist for the loss of her husband through negligence, and she ed a verdict with £800 damages. (*Brit. Med. Record*, Jan. 10, p. 51.)

carbonate of baryta is said to have destroyed life in two cases, in f which only one drachm was taken; but the following case, occurred to Dr. Wilson, shows that this compound is not so poi- as the chloride. A young woman swallowed half a teacupful powdered carbonate, mixed with water, at a time when she had asting twenty-four hours. There was no particular taste. In urs she experienced dimness of sight, double vision, singing in rs, pain in the head, and throbbing temples, with a sensation of distennd weight at the pit of the stom- There was also palpitation of the After a time she complained of a the legs and knees and cramps in ves. She vomited twice, a fluid like and water. The skin was hot and he pulse frequent, full, and hard. symptoms gradually abated, and she red, although the pain in the head omach continued for a long time. *Gaz.*, vol. 14, p. 448.) The car- is used as a poison for rats and mice.

FIG. 13.



Crystals of chloride of barium, magnified 30 diameters.

lysis.—*Chloride of barium* crystal- thin quadrangular plates (Fig. 13); it is soluble in water. 1. lution yields a white precipitate with sulphuric acid or an alka- lphate. This precipitate is insoluble in nitric acid. 2. The pow- salt, burnt on platinum wire in a smokeless flame, imparts to it nish-yellow color. 3. Chlorine may be detected by a solution of of silver.

bonate of baryta is a white insoluble powder. It is entirely dis- with effervescence (carbonic acid) by diluted hydrochloric acid. aporation, it yields crystalline plates of the chloride of barium, may be tested by the processes above mentioned.

NON-METALLIC IRRITANTS.

CHAPTER XXXI.

PHOSPHORUS—SYMPTOMS AND APPEARANCES—CHRONIC POISONING BY THE VAPOR—FATAL DOSE—PERIOD OF DEATH—CHEMICAL ANALYSIS—PHOSPHORUS-PASTE AND MATCHES—RED OR AMORPHOUS PHOSPHORUS—IODINE.

PHOSPHORUS.

CASES of poisoning by phosphorus appear to be much more numerous in France than in England. M. Chevallier has collected seventy-four cases of poisoning by this substance, and in forty-two of these the phosphorus was procured from lucifer matches. Among the cases, twenty-five were the result of accident, twenty-eight involved a criminal charge, and twenty-one were the result of suicide. (*Annales d'Hygiène*, 1857, vol. 2, p. 226.) The cases of poisoning by phosphorus in this country are not very numerous, and they are chiefly referable to accident or suicide. Within a period of five years there were fifteen deaths from phosphorus out of 1620 cases of poisoning. In France, within a period of six years, there were 103 cases of poisoning with phosphorus which gave rise to medico-legal inquiry; and, according to Tardieu, this poison is there often selected for the purposes of suicide or murder.

Phosphorus is seldom used in this country in attempts at murder. The smell and taste as well as its luminosity in the dark commonly reveal its presence. At the Norwich Autumn Assizes, 1871 (*Reg.* v. *Fisher*), a girl of eighteen was convicted of an attempt to poison a family. She put a vermin compound of phosphorus into a teapot containing tea. When hot water was poured on it the smell at once led to suspicion. Phosphorus was found in it, taken from a pot carelessly left about the house. The girl was convicted and sentenced to penal servitude for life. The late Professor Casper, of Berlin, describes a case in which the luminous appearance of the poisoned food led to a suspicion of poisoning with phosphorus, and this was subsequently proved. A woman put a preparation of phosphorus into soup, and gave it to her husband. He ate it in a dark room in the presence of some friends, and they noticed that the warm liquid, as he stirred it, was luminous. (*Vierteljahrsschrift*, July, 1864.) In this way a person may be warned and a life saved. (See *Ann. d'Hyg.*, 1870, vol. 2, p. 203.)

Symptoms.—Phosphorus acts as an irritant poison, but its operation is attended with some uncertainty, according to the state in which it is taken. The symptoms are frequently slow in appearing; it is only after some hours, and sometimes even one or two days, that signs of irritation with convulsions and spasms appear, but when these once come on the case proceeds rapidly to a fatal termination. In the first instance the patient experiences a disagreeable taste resembling garlic, which is peculiar to this poison. An alliaceous or garlic odor may be perceived in the breath. There is an acrid burning sensation in the throat, with intense thirst, severe pain and heat with a pricking sensation in the stomach, followed by distension of the abdomen, and nausea and vomiting continuing until death. The vomited matters are black or of a dark coffee-grounds color; they have an odor of garlic; white vapors, having the peculiar odor of phosphorus, may be seen to proceed from them, and in the dark they may even appear phosphorescent. Purging is among the symptoms, and the motions have been observed to be luminous in the dark. The urine also, passed at an early stage, has been observed to be luminous in the dark. The pulse is small, frequent, and scarcely perceptible. There is great prostration of strength, coldness of the skin, and other symptoms of collapse. The patient may die quietly in a state of coma, or be convulsed before death. Jaundice has been observed among the symptoms. The following cases will illustrate the mode in which this poison acts:

A girl, between two and three years of age, had been caught in the act of sucking and swallowing the heads of lucifer matches. Two days afterwards she appeared unwell; there was some feverish excitement, but no active symptoms. The bowels were open, but the child did not suffer from pain, vomiting, or purging. Five hours after she was first seen, she became violently convulsed, and she died in three hours. On inspection, a quantity of mucus, mixed with blood, of a coffee-grounds color, was found in the stomach. The mucous membrane was very red throughout, and for the space of about two inches it had a florid-red color and was covered with mucus. There were no fewer than ten invaginations in the small intestines (intussusception, *ante*, p. 98) many of which included from two to three inches of intestine, which was inflamed at the invaginated parts. There was no appearance of strangulation, and the bowels were empty. The medical opinion given at the inquest was, that phosphorus, in a finely divided state, was the cause of death, and a verdict was returned accordingly. (*Lancet*, Dec. 1843.) A woman committed suicide by dissolving in vinegar the phosphorus from the ends of some lucifer matches. She swallowed this mixture, and after undergoing the most severe suffering for eight days, she died laboring under symptoms resembling those of hydrophobia. (*Journ. de Chim. Méd.*, 1846, p. 668.)

A woman, æt. 26, swallowed a decoction of lucifer matches in coffee. In an hour an emetic was given to her, and she vomited half a pint of clear, glairy fluid, having the smell of phosphorus and containing particles of blue coloring matter (Prussian blue) derived from the matches. She had no pain in the stomach, and no purging. In four days she appeared to have recovered; but about this time there was bleeding from the nose; she was jaundiced, and blood appeared in the matter vomited. Febrile symptoms set in with purpura, and she died in about a week after taking the poison. (*Ed. Monthly Journal*, October, 1860.) On April 20th, 1861, a girl swallowed a quantity of phosphorus paste. When seen soon afterwards, by Mr. Parsons, of Bridgewater, her lips as well as parts of her dress were smeared with this substance, and there was a strong odor of phosphorus in her breath. Her countenance was tranquil, her pulse regular; there was no sickness or nausea, and she complained of nothing but slight thirst. Her symptoms were so mild that they excited no suspicion that the girl had swallowed the poison. She passed a restless night, and the next day she complained of heat in the mouth and throat, and of a slight sensation of nausea and retching. There was no pain or tenderness in the region of the stomach, the pulse was regular but weak. On the 22d she dressed herself and was able to walk about the ward; she left the hospital and went home, having walked a mile; she had her tea as usual at night, and went to bed. On the following day, the 23d, she complained of pain in her bowels, with sickness and purging. These symptoms became worse. On the 25th there was pain in the bowels, which were tender on pressure and slightly tympanitic. The pulse was intermittent, and the girl was fast sinking. She died on the 26th, having survived the effects of the poison nearly a week, and no well-marked symptoms having set in until *the fifth day*. An inspection of the body was not permitted, and the only fact observed after death was a tendency to rapid putrefaction. The whole of the body became speedily livid, and the finger-nails were blue—a condition noticed by a witness to have existed before death. (For other cases, see *Ann. d'Hyg.*, 1869, vol. 2, p. 397.)

It will be perceived that, in reference to the delay in the appearance of symptoms, their slightness taken as a whole, and the time at which death occurred, this case is similar to one previously related. If it were not for the peculiar character of the circumstantial evidence these cases might easily throw a practitioner off his guard in forming an opinion. The odor of the breath, and the appearance of phosphorus smeared over the dress, first attracted the notice of Mr. Parsons. Other witnesses deposed that whatever deceased touched with her hand seemed to take fire, and that when she drank water to allay her thirst a kind of smoke issued from her mouth. Her hands and dress were luminous in the dark.

A case is reported by Dr. Graff, in which a young woman swallowed the phosphorus obtained from about three hundred matches—equal to rather less than *five grains* of pure phosphorus—and recovered from the effects. The symptoms do not appear to have been very severe—a fact ascribed by the reporter to the phosphorus having been in an intimate and probably insoluble state of combination with other substances in the matches. (Henke, *Zeitschrift*, 1842, vol. 2, p. 283.) Phosphorus in small doses is said to produce strong aphrodisiac effects. This view is borne out by the facts collected by Dr. Hartcop. (See Casper's *Wochenschrift*, 21 Feb., 1846, p. 115.)

Phosphorus Vapor—Chronic Poisoning.—Chronic poisoning by phosphorus is accompanied by nauseous eructations, frequent vomiting, sense of heat in the stomach, purging, straining, pains in the joints, wasting, hectic fever, and disease of the stomach, under which the patient slowly sinks. Some interest is attached to the chronic form of poisoning by phosphorus from the researches of Dr. Strohl and others, on the effects of the *vapor* upon individuals engaged in the manufacture of phosphorus or lucifer matches. It has been remarked that persons thus engaged have suffered from necrosis of the jaw, carious teeth, and abscesses. There has been also marked irritation of the respiratory organs, and bronchitis has frequently shown itself among them. These effects have been attributed to the respiration of the vapors of phosphorus, which are supposed, by becoming acidified, to act chemically upon the bones and exposed portions of the teeth. A good summary of the facts connected with this kind of poisoning, by the late Dr. Beck, will be found in the *American Journal of Medical Sciences* for Oct. 1846, p. 525. (See also *Ann. d'Hygiène*, 1856, vol. 2, p. 5; and 1857, vol. 1, p. 431.) A case, in which pneumonia was considered to have been induced by phosphorus vapor, is reported in the *Med. Gaz.* (vol. 39, p. 210), and another well-marked instance of the serious local and constitutional effects of the acid vapors has been published by Mr. Wright. (*Med. Times*, Dec. 19, 1846, p. 224.) According to M. Dupasquier, phosphorus in vapor has no specific poisonous action. It merely irritates the lining membrane of the bronchial tubes, and this effect is soon lost by habit. When other and more dangerous symptoms supervene, he thinks they should be ascribed to the accidental presence of arsenic in the phosphorus. (*Journal de Pharmacie*, Oct. 1846, p. 284; also, *Gaz. Méd.*, Dec. 5, 1846, p. 946.) This view of M. Dupasquier is not borne out by experience. Numerous facts are now placed on record which show that the vapor of phosphorus alone produces most injurious effects to health.

Since the introduction of allotropic phosphorus for the manufacture of matches, these serious cases of phosphorus disease have not been met with. It is a rare circumstance to hear of an acute case of poisoning by this vapor. Bouchardat mentions the case of a druggist who, while preparing a large quantity of rat-poison from phosphorus in a close room, inhaled the vapors to such a degree that he fainted repeatedly, fell into a state of complete prostration, and died in a week. (*Annuaire de Thérap.*, 1874, p. 109.)

Post-mortem Appearances.—In addition to those already described we may find marks of irritation and inflammation in the stomach and intestines generally. The mucous membrane is inflamed and softened, and in some instances the stomach is contracted.

A boy, æt. 10, took medicinally phosphorus in pills and in an oleaginous mixture for nearly four weeks. He was found lying in a state of stupor, quite insensible, laboring under strong convulsions, hurried breathing, and a small pulse. He died some hours afterwards. The principal appearances in this case were congestion of the brain, a bright vermilion color of the anterior surface of the stomach externally, with softening of the mucous membrane within, and the marks of violent irritation and inflammation of the muscular coats of the large intestines. The quantity of phosphorus taken is not stated; it was given in divided doses, and none had been taken for ten days previously to death; nevertheless death was ascribed to the long-continued use of the substance. The stomach contained two ounces of a dark-brown liquid, and a large quantity of mucus. (*Lancet*, Sept. 14, 1844.)

Inflammation of the stomach and bowels proceeding to gangrene is a common result of the action of phosphorus. M. Worbe found the stomach perforated in three places in a dog which had been poisoned by a solution of phosphorus in oil.

A man, æt. 50, took a quantity of phosphorus paste used for destroying vermin. He was seen in his usual health at twelve o'clock P.M., and was found dead in a field the following morning. On inspection, it was observed that there was great muscular rigidity. The membranes of the brain were congested, and there was serous effusion between the arachnoid and pia mater. The substance of the brain was also congested. The heart was flaccid and nearly empty. The mucous membrane of the stomach, gullet, and small intestines was very red, and there were patches in which the membrane was destroyed. On opening the stomach a white smoke escaped, accompanied by a strong smell of garlic. It contained a tablespoonful of viscid greenish matter, from which particles of phosphorus with some Prussian blue (used as a coloring for the poison), subsided on standing. (Dr. Bingley, *Lancet*, June 13, 1857, p. 600.) The late Mr. Herapath states that, in a case which he examined, he found, besides inflammation of the stomach, the mucous membrane raised in small bladders or vesications. As the body was not examined until twenty-three days after death, this was no doubt a change produced by putrefaction. Such a blistered appearance is frequently seen in cadaveric inspections, and has not been observed in recent cases of poisoning by phosphorus. Schuchardt describes, among the appearances, fluidity of the blood, which is of a dark color, and does not become red on exposure to the air. Another remarkable appearance frequently met with is a fatty degeneration of the liver and other soft organs. Ecchymoses are also found on the skin and on the surface of various organs of the chest and abdomen. (*Brit. and For. Med. Rev.*, 1857, vol. 9, p. 506. *Journal de Chimie Médicale*, 1857, p. 84.)

In two cases of acute poisoning with phosphorus, communicated to me by the late Dr. W. D. Moore, one proved fatal in seventy-two, and the other in eighty-eight hours. The symptoms and appearances were similar in those already described. Fatty degeneration of the liver and other organs was especially marked. (See *Medical Press*, Nov. 15, 1865, p. 434.) In a case which occurred to Dr. Anderson, a child aged one year and eight months had sucked the heads off about twenty phosphorus matches before it was detected. No symptoms appeared until the second day, when the child was drowsy and slept for twenty hours. Castor-oil and oil of turpentine were given. On the fourth day it vomited, the skin was hot, tongue dry, there was great thirst, with a quick pulse and cold extremities. On the sixth day there was much vomiting of a matter like coffee-grounds (altered blood). There was severe pain in the stomach—the child became unconscious and gradually sank, dying on the seventh day after taking the poison. There was no purging, but the motions were passed involuntarily, containing coagulated blood. An alliaceous odor was perceived in the breath during the progress of the case, and the body had a yellowish (icteric) tint. On inspection there was marked general ecchymosis. The liver was enlarged, and of a yellowish color, undergoing fatty degeneration. The lining membrane of the stomach was injected, and it contained a dark bloody fluid. There was no odor of phosphorus, and the contents were not luminous in the dark. Phosphorus could not be detected by Mitscherlich's process. (*Lancet*, 1871, vol. 2, p. 189.)

In a case elsewhere described, p. 273, which proved fatal in a week, there was no inflammation, ulceration, or softening of the mouth, gullet, stomach, or small intestines. There was a red patch in the cæcum, and another in the colon (the large intestines). The contents of the stomach and intestines had a coffee-grounds color, like the liquid found in hæmatemesis (vomiting of blood). The brain was slightly congested. There were bloody effusions in the chest and abdomen, and an apoplectic condition of the soft organs. The vomited matters, when shaken in the dark, were luminous, and phosphorus was separated from them by sulphide of carbon. In a case which I examined in 1867, that of a girl, æt. 13, who died on the sixth day after taking phosphorus paste beaten up with egg, there were the usual symptoms, with severe paroxysms of vomiting and pain. The matters first vomited were observed to be luminous in the dark. There were numerous ecchymosed patches in the cellular tissue of the skin of the abdomen over the rectus muscle; these were also seen on the chest and on the diaphragm. The stomach contained a dark-colored thick fluid like altered blood; the coats were not inflamed; the surface of the inner coat was covered with a brownish-colored mucus which had no odor of phosphorus. At the greater curvature the surface was dotted over with numerous small dark particles, consisting of coagula of altered blood, adhering to the membrane, but easily removed from it. They had the appearance of effused coagula of blood in petechial spots. The contents of the stomach owed their color to these little masses of blood being diffused through them. The duodenum contained a similar liquid. The intestines presented no abnormal appearance. The liver was in an advanced state of fatty degeneration. This condition of the liver has occurred so frequently in cases of phosphorus poisoning, that it may now be regarded as one of the characteristic appearances. (*Guy's Hospital Reports*, 1868, p. 242.) M. Tardieu has met with this fatty degeneration in poisoning with phosphorus, not only in the liver, but in the heart and kidneys. (*Étude Méd.-Lég. sur l'Empoisonnement*, 1867, p. 441.) In this work the reader will find a complete history of this form of poisoning.

In an interesting case recorded by Dr. Habershon (*Med.-Chir. Trans.*, 1867, vol. 50), in which a woman died on the fifth day, the symptoms and appearances were similar to those above described. The phosphorus was taken in the form of paste, and it is supposed in a dose of from three to four grains. There was much ecchymosis in patches, in and about the cellular tissue of the abdomen and chest. There was fatty degeneration of the liver and kidneys. The stomach contained a large quantity of fluid like soot and water, and was covered with a tenacious bloody mucus. There was some congestion in the mucous membrane, but there was much redness with ecchymosis in the small intestines.

(For further information on this subject, see *Die acute Phosphor-Vergiftung von Munk und Leyden*, Berlin, 1865. Horn's *Vierteljahrsschrift*, 1866, vol. 1, p. 271, and Wiggers and Husemann's *Jahresbericht*, for 1872, p. 472.) The viscera, and even the flesh of animals recently poisoned by phosphorus, have the odor of garlic, and appear luminous in the dark. (Galtier, *Toxicologie*, vol. 1, p. 184.) Mr. Clowes informed me, that in examining some fowls which had been poisoned by phosphorus, he was struck with the strong odor of this substance on opening the gizzards, and with the appearance of a fine white fume, which was luminous when observed in a dark room. In the case of a woman who died while taking phosphorus medicinally, it was remarked that the whole of the viscera of the body were luminous in the dark; thus indicating the extensive diffusion of the poison by absorption. (Casper's *Wochenschrift*, Feb. 21 and 28, 1846, pp. 115, 135.) For a further account of the appearances, see *Chemist*, Jan. 1856, p. 244.

That this poison is absorbed and diffused through the body is established by the luminosity of the viscera, which has been frequently observed. Vauquelin, after having exposed himself to the vapor of phosphorus, observed that the urine which he passed soon afterwards was phosphorescent, and M. Chevallier states, on the authority of a phosphorus manufacturer, that on many occasions the men who were employed in his establishment, and who were in the habit of breathing phosphorus vapor, passed phosphorescent urine. (*Annales d'Hygiène*, 1857, vol. 2, p. 214.) It is not improbable that this substance may be eliminated by the lungs, and that the breath of persons poisoned by phosphorus may be luminous in the dark.

Fatal Dose.—That phosphorus is a powerful poison, is proved by two cases quoted by Sir R. Christison. In one, death was caused by a grain and a half in twelve days; in the other, by two grains in about eight days. It has been supposed to operate as a poison only by becoming converted into phosphorous acid; but although this conversion takes place, it is probable that phosphorus passes directly into the blood, since the urine first voided has been observed to be luminous in the dark; hence it is itself probably a blood poison. The production of phosphorous acid, by its oxidation, may account for the erosions met with in the stomach and bowels, as also for the rapid disappearance of the poison from the body. The fatal dose is liable to vary according to many circumstances. Galtier states that it is comprised between three-quarters of a grain, and two grains, and that even a third of a grain has destroyed life; while persons have recovered, as in one instance referred to, from a dose of *five* grains (p. 274). In the case of a man, æt. 27, reported by Worbe, and quoted by Orfila, the ascertained fatal dose was less than *a grain and a half*. The phosphorus was melted in hot water, and thus swallowed. Three days previously he had taken less than half a grain (three centigrammes) without ill effects. The patient suffered from all the symptoms of phosphorus poisoning, and died in twelve days. It is worthy of remark, however, that no active symptoms showed themselves for several hours. (*Toxicologie*, vol. 1, p. 55.)

Dr. Hartcop mentions that an apothecary took, by way of experiment, one grain; on the next day two grains, and on the third day three grains of phosphorus, mixed with sugar. He was then seized with inflammation of the stomach and bowels, and died in spite of every attempt to save him. (Casper's *Wochenschrift*, 1846, p. 117.) M. Chevallier refers to a case in which a dose of 2.3 grains proved fatal, and two other cases in each of which a dose of 4.6 grains destroyed life. The same writer quotes, on the authority of Löbenstein Löbel, of Jena, the case of a lunatic who died from a dose of one-eighth of a grain. (*Ann. d'Hyg.*, 1857, vol. 1, p. 422.) Excepting this, the smallest fatal dose which I have met with, is in a case quoted by Galtier. A woman, æt. 52, took in divided doses, in four days, about six centigrammes, or less than *one grain*, of phosphorus dissolved. The largest dose taken at once, *i. e.*, on the fourth day, is stated to have been three centigrammes (0.462 grain), or less than half a grain. Symptoms of pain and irritation appeared, and the patient died in three days. The gullet, stomach, and small intestines were found much inflamed. (*Toxicologie*, vol. 1, p. 87.) When the phosphorus is dissolved in any liquid, or when it is very finely divided, as in phosphorus paste or in lucifer matches, its action is then more powerful, as it is in a state well fitted for absorption.

When given medicinally dissolved in ether or oil, it has been known to produce alarming symptoms, and to endanger life. In one case, severe vomiting was induced, the vomited matters having a glairy, bilious appearance, and this continued for sixty hours. Jaundice set in, and the urine was tinged with bile. (Bouchardat, *Ann. de Thérap.*, 1872, p. 80.) It is now regarded by French practitioners as a dangerous medicine, and one requiring special precautions in its employment for medicinal purposes.

Period at which Death takes place.—This has varied greatly, in the cases hitherto observed, from a few hours to a week. In a case related by Orfila death took place in four hours. In another, also related by him, death occurred only after seventeen days. Dr. Habershon quotes a case which is said to have proved fatal in *half an hour*. (*Med.-Chir. Trans.*, 1867, vol. 50.) This is the shortest period recorded. In general, several days elapse before a fatal result occurs, and during this time the patient undergoes much suffering. This was observed in a young woman who swallowed a quantity of phosphorus paste intended for poisoning rats. She did not die until *the fifth day*. (*Journal de Chimie Méd.*, 1845, p. 580.)

Treatment.—This may consist in the administration of emetics, and of albuminous or mucilaginous drinks holding hydrate of magnesia suspended, as well as in the free use of emetics and purgatives. When the symptoms have once manifested themselves, it is difficult to arrest their progress, and there is no known antidote to this poison when it has once entered into the blood. Linseed oil has been recommended, but on no sufficient grounds. Oil of turpentine has been used and regarded as an antidote after giving an emetic, but as it partially dissolves phosphorus, it might bring the poison into a state better fitted for absorption, and thus do mischief. (Bouchardat, *Ann. de Thérap.*, 1873, p. 100.)

CHEMICAL ANALYSIS.

Phosphorus is a solid of waxy consistency, having a peculiar odor and a taste resembling that of garlic. The odor and taste prevent it from being criminally employed as a poison, and lead to its detection in articles of food. It evolves a white vapor in daylight, and a faint bluish luminosity in the dark. It melts and takes fire at a temperature of about 113°, burning with a bright yellow flame, and producing thick white acid vapors by combustion. It is not soluble in water, but water in which it has been preserved or washed acquires poisonous properties by reason of the phosphorous acid formed. (*Ann. d'Hyg.*, 1857, vol. 1, p. 423.) Phosphorus is dissolved by alcohol, ether, chloroform, and the oils, but especially by sulphide of carbon.

Organic Mixtures.—The smell which phosphorus imparts to solid and liquid organic substances is remarkably characteristic. When it has been taken in a solid form, it may be separated as a sediment in fine particles by washing the contents of the stomach in water.

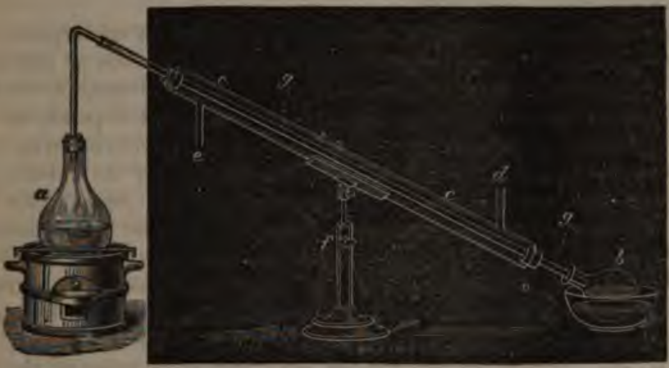
They may be melted under water into one mass, either by plunging the tube containing them into hot water, or by pouring hot water upon them in a conical glass. If a portion of the organic liquid is evaporated to dryness in the dark, the particles of phosphorus will be easily recognized by their luminosity, as well as by their combustion when the surface on which the material is spread is further heated.

Phosphorus is very soluble in sulphide of carbon, and it may be separated from many organic matters by digestion with this liquid. It is thus procured from flour and phosphorus paste, or from the residue of the contents of the stomach after washing and decantation. On the spontaneous evaporation of the sulphide, decanted from the organic liquid or solid, the phosphorus may be procured in small globules or beads. These are ignited when touched with a hot wire. A portion of the solution poured upon thin paper, ignites spontaneously when dry, and burns with the well-known flame.

If the phosphorus is in a state of solution, or is in too small quantity to be dissolved out of the material by sulphide of carbon, its presence may be indicated by distilling the liquid containing it in a perfectly dark chamber.

This process, which was first suggested by Mitscherlich, for the detection of small quantities of phosphorus, removes any difficulty respecting the presence or absence of the poison. Mitscherlich adds to the organic substance a sufficiency of water to make it quite fluid, and a small quantity of sulphuric acid to neutralize any ammonia, and raise the boiling-point. The mixture is placed in a capacious flask, connected with a long glass condensing-tube, kept cool by a stream of cold water. The tube is fitted into a receiver. The suspected liquid is distilled in the dark, and if a minute trace of phosphorus be present, *i. e.*, the 100,000th part, or according to De Vrij, the 2,000,000th, the fact will be made evident by a luminous appearance in the upper part of the tube, at each successive condensation of the vapors. The black space represents a dark chamber, on the outside of which the flask *a*, containing the liquid for distillation, is placed; *b*, a flask acting as a receiver for the condensed aqueous vapor; *c c*, glass condensing-tube kept filled with cold water entering at *d* and passing out at *e*; *f* is a stand; *g g* represent the small tube through which the phosphorus vapor passes, mixed with aqueous vapor. The luminosity of the phosphorus vapor is seen at the point at which the aqueous vapor is condensed, generally between *g* and *d*. For the success of this experiment there must be the most absolute darkness. The bend of the tube of the flask *a* should be provided with a metallic cowl. If ether or alcohol should be present, the vapors of these liquids should be distilled over first. In the receiver in which the vapor of the distilled liquid is condensed, phosphorous acid or phosphoric acid may be discovered by the usual tests. So delicate is this process of distillation and condensation, that in one experiment with the head of a single lucifer-match, I observed that the luminosity continued for half an hour in the condensing-tube. The most absolute darkness is required for the success of this experiment.

FIG. 14.



Apparatus for detecting phosphorus by distillation.

If the person has survived several days it is not likely that any free phosphorus will remain in the stomach or its contents. None was found in the contents of the stomach or in the fatty liver of the girl who died on the sixth day (page 277), but the distillation process succeeded with the broken top of a pot which had held the phosphorus paste, although this was empty and had been thrown into a tub of water. In Dr. Habershon's case of death on the fifth day, none was found by Dr. Stevenson in the stomach or contents. The phosphorus in these cases is oxidized rapidly, and thus, like other poisons, it may disappear from the body. Under these circumstances it may, according to some authorities, be still discovered as phosphoric acid. M. Mialhe has given an elaborate report on a case in which the symptoms and appearances were those of phosphorus poisoning, the girl dying on the fifth day. Mitscherlich's process failed to show any free phosphorus. Eight weeks afterwards portions of the viscera were examined by MM. Tardieu and Roussin. They found in the intestines and on the liver groups of small crystals of ammonio-phosphate of magnesia, and in the fluid contents an acid liquid having the properties of phosphoric acid. (*Ann. d'Hyg.*, 1869, vol. 1, p. 134.) These crystals, it may be observed, are frequently found as a result of decomposition in the stomach or on the surfaces of the liver, kidneys, and other organs, without reference to poisoning by phosphorus. As the phosphates are naturally present in the secretions, which are generally acid, it would be difficult to satisfy an English court of law that their presence proved poisoning by phosphorus, unless the symptoms, appearances, and circumstantial evidence were so strong that chemical evidence was scarcely necessary.

Non-detection of Phosphorus.—Phosphorus readily undergoes oxidation in the body, and is thus converted into phosphorous or phosphoric acid. M. Blondlot has suggested a process for its detection when this conversion into phosphoric acid has taken place. It depends on a peculiar green color which the lower oxides of phosphorus give to the flame of nascent hydrogen when burnt. (See *Journal de Chimie*, 1862, p. 528; also a paper by Dr. Ludwig, in the same journal, for 1863, p. 581.) The late Mr. Herapath suggested this some years since as a method of detecting phosphorus thus changed in the body, and he employed it in one medico-legal case. Mr. Barrett has lately shown, by a variety of experiments, that the flame of pure hydrogen is rendered of a vivid green by an infinitesimal trace of phosphorus (*Nature*, April, 1872, p. 483), but as phosphates are constituents of most of the solids and fluids of the body, this mode of testing would hardly be applicable to medico-legal purposes. It requires for medigo-legal application materials of absolute purity for procuring hydrogen as well as a pure atmosphere and perfect darkness.

This subject has been lately examined by M. Lefort (*Ann. d'Hyg.*, 1874, vol. 1, p. 405). He has shown that phosphorus is very likely to disappear as a result of oxidation in the living and dead body, and that after two or three days' survivorship, or two or three weeks' interment, none may be found. The late Mr. Herapath failed to find any trace in a body examined on the twenty-third day after death. In the following case, which occurred to Dr. Neumann, none was found in the body of a man who died in two days, the body having been exhumed after fourteen days' burial. The chemical evidence, however, was supplied from another source. A shepherd, after having eaten some beetroot soup, vomited several times, complained of thirst, intense pain in the abdomen, and died after two days' continuous suffering. His dog, which had eaten some of the food, became unwell, and died in two hours. The man lived unhappily with his wife, and, from some suspicion as to the cause of death, the body of the deceased, as well as that of the dog, was ordered to be disinterred and examined. As the bodies had been buried *fourteen* days, and the weather was warm, they were in an advanced state of decomposition. It was impossible to draw any inference of poisoning from the appearances of the viscera. A portion of the soup of which the deceased and his dog had eaten was procured and submitted for examination. A small quantity was spread on an iron plate dried and heated to a moderate temperature.

Portions immediately burnt with a yellow light and a thick white smoke. In addition to this the soup had the smell of phosphorus, and when warmed was luminous in the dark. (Casper's *Wochenschrift*, May 31, 1854.)

M. Lefort relates two cases, in one of which the patient survived three days and the other seven, but no trace of free phosphorus could be found in the bodies. In one of these, however, there was a general steatosis or fatty degeneration of the organs. He properly objects to any opinion of phosphorus poisoning being based on the detection of phosphoric acid or the phosphates. These are naturally contained in the organs and fluids of the body, and it would be unsafe to infer that under any circumstances their presence furnished a proof of poisoning by phosphorus. In the absence of any traces of free phosphorus the fact of poisoning must be proved by general and pathological evidence.

Phosphorus Matches.—It is now rare to find the waxy or poisonous form of phosphorus in matches. The proportion contained in the heads of the matches was found to vary greatly. The dry composition was said to contain as much as one-fourth of its weight. The presence of ordinary phosphorus may be detected in them by soaking the heads in sulphide of carbon, or, better, by distillation (p. 281).

When the phosphorus has been scraped from the tips of matches it may be oxidized and lost, but as it is usually colored with vermilion, Prussian blue, or some other coloring matter, these mineral substances may be found in the washed sediment of the contents of the stomach. On the non-discovery of free phosphorus in the body, these coloring matters, if present, serve to indicate the form in which the poison has been taken or administered. In a case which occurred to Tardieu and Roussin sulphur was found as well as phosphorus. (*Ann. d'Hyg.*, 1868, vol. 1, p. 117.)

Phosphorus Paste.—This consists of phosphorus in a finely divided state mixed with a farinaceous paste, and sometimes colored with Prussian blue. The sample appears white until exposed to the air. The substance has the powerful odor of phsphorus, it fumes in the air, giving off the usual white vapors of phosphorous acid. When spread in a thin layer on a sheet of mica and heated, the particles of phosphorus burn with bright scintillations, and the farinaceous matter is carbonized. The paste is said to contain one-eightieth of its weight of phosphorus. (*Ann. d'Hyg.*, 1869, vol. 2, p. 396.) The phosphorus contained in it may be converted into phosphoric acid by boiling it with nitric acid slightly diluted, or it may be dissolved out of the paste by sulphide of carbon. The farinaceous portion of the compound may be known by the addition of iodine and the application of the microscope. This paste is luminous in the dark, giving off a visible phosphorescent vapor. It is colorless when not in contact with air, so that the blue color from Prussian blue may not be seen when the stomach is first opened. This effect of color should be borne in mind. The vomited matter in poisoning by phosphorus, as well as the contents of the stomach after death, may be blue. If the blue color depends on Prussian blue, it will be entirely destroyed by the ammonia of putrefaction. According to one formula, this substance consists of one drachm of phosphorus (finely divided by melting it in rectified spirit), five ounces of flour, and an ounce and a half of brown sugar, made into paste with a little water. (*Pharm. Journ.*, 1852–3, p. 402.)

Red or Allotropic Phosphorus.—The remarkable substance, known under the name of allotropic or amorphous phosphorus, is not possessed of poisonous properties. This fact, long since announced by Liebig (*Letters on Chemistry*, p. 165), has been confirmed by experiments at the Veterinary College at Alfort. (*Ann. d'Hyg.*, 1857, vol. 1, p. 432.) Common phosphorus is poisonous in doses varying from one to three grains, while allotropic phosphorus has been given to animals in doses of thirty grains without causing symptoms of poisoning. This kind of phosphorus, by reason of its being generally in a fine powder, is in a state more favorable for acting as a poison than common phosphorus; and yet, owing probably to its insolubility, it is inert. M. Bussy in 1850, and M. de Vrij in 1851, proved that a dog might take with impunity thirty grains. Orfila and Rigault have given it to animals in doses amounting to some ounces, over a period of twelve days, without producing any noxious effects. (See *Annuaire de Thérapeutique*, 1855, p. 103.) That it does not act as a poison in the human body, appears to be established by the facts of a case reported in the *Edinburgh Monthly Journal*, for October, 1860. A woman, æt. 26, swallowed the composition scraped from a number of lucifer matches; it turned out that these were made with allotropic phosphorus. She suffered no inconvenience. She procured other matches of common phosphorus, took a decoction of them in coffee, and died from the effects.

Analysis.—Allotropic phosphorus is easily recognized by heating it, or any mixture containing it, to about 500°, when it burns like common phosphorus, and yields similar products. It is insoluble in all liquids, and by its insolubility in sulphide of carbon, it is distinguished and separated from common phosphorus. It has no odor or taste, and is not luminous in the dark, unless it contains common phosphorus. In any analysis for phosphorus, we must take care to exclude it by employing sulphide of carbon as a solvent for the common or poisonous form. (The reader will find a full account of the comparative effects of the common and allotropic phosphorus by M. Chevallier, in the *Annales d'Hygiène*, 1856, vol. 1, p. 374. See also the same journal, 1859, vol. 2, p. 370, and Casper's *Vierteljahrsschrift*, 1860, vol. 2, p. 185.)

IODINE.

Symptoms.—From experiments on animals, as well as from observation of its effects on man, iodine has a strong local action as an irritant on the stomach and bowels. In large doses, it occasions a burning heat in the throat, severe pain in the abdomen, with vomiting and purging; the vomited matters having the peculiar marine odor of iodine, and being of a yellow color, except when any farinaceous food has been taken, in which case they are blue, or even black. The fecal matters may also contain iodine if the poison has been taken in the solid state. Besides these symptoms, there is great thirst, with anxiety, headache, giddiness, trembling and convulsive movements of the limbs, and fainting; these last symptoms indicating that the poison has become absorbed. When taken for some time in small doses, it gives rise to salivation, vomiting and purging, pain in the stomach, and cramps; the pulse is small and frequent; there is a general wasting of the body; and it has been observed that, in the form of chronic poisoning, certain glands are liable to become affected and diminished by absorption—the breasts in the female, and the testicles in the male. Iodine produces these secondary effects (iodism), whether it is taken internally or applied externally. A woman swallowed, by mistake, one drachm of iodine dissolved in an ounce of alcohol. When seen afterwards, she complained of a violent pain in the throat or stomach, followed by retching and slight vomiting; pulse rapid and full; eyes prominent and suffused. Vomiting, promoted by diluents, brought no relief to the symptoms. She became much depressed, and died on the following day. There was no examination of the body (*Prov. Journ.*, June 30, 1847, p. 356). For a case of recovery from half a drachm, see *Med. Times and Gaz.*, Dec. 28, 1861, p. 659.

Iodine is rarely used as a poison. In May, 1864, an attempt was made by a woman to poison a fellow-servant by mixing tincture of iodine with food in a plate. The remarkable discoloration of the farinaceous food, which it produced, led to suspicion and prevented any ill effects from following. Iodine gives a blue, green, or dark color to most organic liquids, and imparts to them a most peculiar marine odor. It stains the skin and other organic substances yellow, the color being removed by an alkali. When in strong solution, it is corrosive and destroys the parts which it touches; in this state it has been maliciously employed for throwing on the person.

Appearances.—As this is an irritant as well as a corrosive poison, the lining membrane of the gullet, stomach, and intestines is found inflamed and excoriated. In one instance the mucous membrane, near the pylorus, was corroded and detached in a space of two or three inches.

Analysis.—The odor is in general sufficient to identify it. This may be concealed by alkalies or alkaline substances. When heated, it sublimes in a purple vapor. The addition of a cold solution of starch produces a blue color, but many substances prevent this reaction. It is very soluble in sulphide of carbon, forming a rich pink solution. The sulphide has the property of removing it from water or organic liquids in which it is dissolved. It may thus be separated for chemical examination by decanting the watery liquid from the sulphide, which, on evaporation, leaves the iodine in crystals. From organic liquids it may be separated by simple distillation.

METALLIC IRRITANTS.

CHAPTER XXXII.

WHITE AND COLORED ARSENIC—ARSENIOUS ACID—TASTE—WEIGHT AND SOLUBILITY—NOT A CORROSIVE—SYMPTOMS IN CASES OF ACUTE POISONING—THEIR COMMENCEMENT AND PROGRESS—NEUROTIC SYMPTOMS—CHRONIC OR SLOW POISONING—DIAGNOSIS—ARSENIC NOT AN ACCUMULATIVE POISON.

WHITE ARSENIC—ARSENIOUS ACID.

THE term WHITE ARSENIC is commonly applied to the arsenious acid of chemists. It is seen under the form of a white powder, visibly crystalline in a strong light, or when viewed with a lens. It is also met with, but more rarely, in opaque, brittle, heavy white masses, resembling enamel. It is called an acid, from its power of combining with alkalies, but it possesses a feeble acid reaction when dissolved in water. It is often described as having an acrid taste, but this does not appear to be correct; a small quantity of it has certainly no appreciable taste, a fact which may be established by direct experiment, and might be inferred from its sparing solubility in liquids. It would appear, from numerous cases on record, that it has been unconsciously taken in fatal quantities, in all descriptions of food, without exciting the least sensation on the tongue. Most of those persons who have been criminally or accidentally destroyed by arsenic have not been aware of any taste in taking the poisoned substance. In cases in which the powder has been taken in *large* quantity, it is described as having had a *roughish* taste. (See the cases of three children, *Guy's Hosp. Rep.*, 1865, p. 282.) There is certainly no ground for assigning to it an acrid taste. In some instances it has produced a decided and persistent impression on the tongue, compared by some to the taste of common salt.

Arsenic is still frequently used as a poison in *rural districts.* From the neglect of common precautions the *accidents* which occur are still numerous. The number of deaths from this poison reported to have occurred in England and Wales in five years, 1863–7, was eighty-three. In India it appears to be a favorite poison. Dr. B. Brown informs me that in the Punjab alone, from 1861 to 1873, the cases of poisoning with arsenic were 1022.

As it is sold to the public in small quantities, it should be mixed either with the 16th part of its weight of soot, which gives to it a grayish color; or the 32d part of its weight of indigo, and then it is blue. Both of these colors are rendered much deeper when the powder is wetted, so that the sooty compound is then nearly black. Sometimes, in place of indigo, artificial ultramarine is employed as a coloring.

The act regarding the coloring of arsenic (14 Vic., c. 13, s. 3) is frequently evaded. It is sometimes sold uncolored under the name of *mercury*. The vomited matters in case of poisoning by arsenic may therefore be blue or black, or the admixture of bile may render them of a deep green color. In a case of arsenical poisoning, communicated to me by Dr. Maclagan, the *blue* vomiting at first completely misled those who were called to render assistance. As soot and indigo are both insoluble in water, these substances will be slowly deposited from the vomited matters by subsidence, and the color given by blood or bile may then become perceptible.

A medical witness may be asked the weight of common or familar measures of arsenic in powder. It may therefore be stated that a teaspoonful of finely powdered white arsenic weighs 150 grains; a tablespoonful weighs 350 grains; and a pinch, or the quantity taken up between the finger and thumb of an adult, weighs 17 grains. The weights here given are the results of actual experiments, but they are of course liable to vary.

Solubility of Arsenic.—The *solubility* of this substance in liquids is a frequent question on trials. The action of water is materially influenced by circumstances. I have found that hot water, cooling from 212° on the poison in powder, dissolves about the 400th part of its weight. This is in the proportion of nearly one grain and a quarter of white arsenic to about one fluid ounce of water. Water boiled for an hour on the poison and allowed to cool, holds dissolved the 40th part of its weight, or about twelve grains to one ounce. Cold water allowed to stand for many hours on the poison, does not dissolve more than from the 1000th to the 500th part of its weight, *i. e.*, one half grain of arsenic to nearly one fluid ounce of water. The presence of organic matter in a liquid renders the poison much less soluble. Thus, hot tea with milk and sugar, and cold porter, did not take up more than about half a grain to the ounce; while hot coffee and cold brandy did not dissolve more than one grain to the fluid ounce. (*Guy's Hosp. Rep.*, vol. 4, p. 103.) Arsenic is dissolved by most organic liquids, as milk, coffee, tea, wine, brandy, whisky, and even oil. Although it is less soluble in these liquids than in distilled water, it is, nevertheless, taken up in sufficient quantity to occasion serious accidents, and even to destroy life. Any alkali or alkaline carbonate dissolved in the liquid, greatly increases its solubility. Liquids which are viscid or mucilaginous, such as gruel, arrowroot, cocoa, or syrup, may mechanically suspend the poison in almost any quantity, but in these cases it cannot be said to be dissolved. A medical witness must always take care to draw a distinction between an actual solution and a mechanical suspension of the poison in a viscid liquid, especially when it is necessary to determine whether the quantity taken was sufficient to kill. The case of Madeline Smith (*ante*, p. 161) involved a point of this nature. A doubt was raised whether eighty grains of arsenic (found in the stomach of the deceased) could have been taken by him unknowingly; and it was considered difficult to suggest a vehicle in which so large a dose could have been secretly administered. There is no doubt that this, or even a still larger dose of powdered arsenic, might be secretly administered in such liquids as gruel or cocoa.

Arsenic not a Corrosive.—Arsenic is an irritant poison; it has no decided chemical or corrosive action on the animal tissues, and the changes met with in the stomach and bowels of a person poisoned by it, are referable to the effects of inflammation. I have not found that arsenic produces any chemical changes on dead mucous membrane. Nevertheless, one instance at least is on record, in which it is alleged to have exerted a corrosive action as a poison. A man named Soufflard, on being condemned to death, swallowed three drachms of arsenious acid in powder; he vomited almost immediately. When seen shortly afterwards the lower lip was strongly cauterized (*fortement cautérisée*); the mucous membrane was white, fissured, and the slightest touch produced excessive pain. The tongue was swollen, and the patient complained of a horrible taste in his mouth and fauces. After death, which occurred in thirteen hours, the membrane of the tongue was found destroyed. (Flandin, *Op. cit.*, vol. 1, p. 495.) Arsenic was detected in the stomach, the mucous coat of which was destroyed, or reduced to a gelatinous pulp; but it is not stated whether it was mixed with corrosive sublimate or any other poison. This action on the mouth is very similar to that produced by corrosive sublimate. According to the reporter of this case, arsenic in a large dose corrodes and destroys the tissues with which it comes in contact; in his opinion it acts like an acid or a caustic substance (vol. 1, p. 557).

Symptoms—Acute Poisoning.—These will vary according to the form and dose in which the poison has been administered. The *time* at which they come on is generally in from half an hour to an hour after the poison has been swallowed. This is the average period. I have known them to appear in a quarter of an hour. Sir R. Christison mentions an instance in which the symptoms began in eight minutes. In the case of Lofthouse, tried at the York Lent Assizes, 1835, the symptoms were proved to have attacked the deceased while he was in the act of eating a cake in which the poison was administered. On the other hand, in an instance communicated to me by Mr. Todd, where one drachm had been taken on an empty stomach, no symptoms appeared for two hours; in one reported by Orfila, the symptoms did not show themselves for five hours; and in another, which occurred to Dr. Lachèse, where a large dose was taken, the symptoms did not appear for seven hours. (*Ann. d'Hyg.*, 1837, vol. 1, p. 244.) Dr. Thompson, of Liverpool, states that he met with a case in which from thirty to forty grains of arsenious acid, and the same quantity of chrome yellow, were taken. Symptoms of poisoning did not appear until five or six hours afterwards. (*Med.-Chir. Review*, 1854, p. 294.) There may be every variety between these extremes. In the cases of three children, elsewhere reported, who took at the same time a quantity of arsenic mixed with flour and sugar, the symptoms appeared in one in two hours, and in the other two in five hours. (*Guy's Hosp. Rep.*, 1865, p. 282.) A remarkable instance occurred to M. Tonnelier, in which the poison was taken by a young woman at eleven o'clock in the morning, and no well-marked symptoms occurred for *eight hours*—there was then violent vomiting. After death a cyst, formed of mucous membrane, and containing arsenic, was found in the stomach; the poison having thus become sheathed over. (Flandin, vol. 1, p. 535.) In a case communicated by Mr. Clegg to the *Medical Times* (Oct. 21, 1848), symptoms of violent irritation did not show themselves until twenty-three hours after the poison had been taken, and within about half an hour of the death of the patient. The girl was once sick shortly after having taken the poison, but the first decided symptoms were those of narcotism. The girl was a confirmed opium-eater, and this habit may in some measure have influenced the operation of the poison. From a case communicated to the *Medical Gazette*, by the late Dr. W. Burke Ryan (vol. 47, p. 722), it appears that the active symptoms of irritation which commonly attend arsenical poisoning, may not show themselves until after the lapse of *nine hours* from the time at which the poison has been swallowed. With the exception of the case above referred to, in which the interval was *ten hours*, this is, I believe, the longest case of protraction on record. In other instances there have been long intermissions. In all cases in which arsenic enters the system from without, as by its application to the skin or to ulcerated or diseased surfaces, the symptoms are rarely manifested until after the lapse of several hours.

Their Nature.—In an *acute* case of poisoning by arsenic the person first experiences faintness, depression, nausea, and sickness, followed by an intense burning pain in the region of the stomach, increased by pressure. The pain in the abdomen becomes more and more severe; and there is violent vomiting of a brown turbid matter, mixed with mucus, and sometimes streaked with blood. These symptoms are followed by purging, which is more or less violent; and this is accompanied by severe cramps in the calves of the legs. The matters discharged from the stomach and bowels have had in some instances a yellowish color, as it was supposed, from a partial conversion of the poison to sulphuret; but more probably from an admixture of bile. The vomited matters are in some cases colored by blood or a mixture of blood and bile; they then present various shades of brown or olive green. The indigo used in coloring arsenic may give to them a blue color, or if mixed with bile a green tint. The sooty arsenic renders them black. In other cases the vomited matters present a milky white appearance, consisting of flakes of mucus mixed with portions of white arsenic. The nature of the arsenic taken may be inferred from the color of the matter vomited. In the case of L'Angelier (*Reg.* v. *Smith*, Edinburgh, 1857), a witness deposed that the matter vomited by deceased, in the first stage of his illness, was a greenish substance of about the thickness of gruel. (*Irvine's Report*, p. 30.) Such would be the appearance produced by a mixture of blue arsenic and bile. Blue arsenic was in this case traced to the possession of the prisoner; but from an altered state of the bile there may be green vomiting even when white arsenic has been taken.

The vomiting is in general violent and incessant, and excited by any substance taken into the stomach. It brings no relief. There is tenesmus (straining), and the discharges by the bowels are frequently tinged with blood. There is a sense of constriction, with a feeling of dryness or burning heat in the throat, commonly accompanied by the most intense thirst. The pulse is small, very frequent, and irregular; sometimes wholly imperceptible. The skin is cold and clammy in the stage of collapse; at other times it is very hot, or there are rapid alternations of heat and cold. There is great restlessness. The breathing is painful from the tender state of the abdomen. Before death coma sometimes supervenes, with paralysis, tetanic convulsions, or spasms in the muscles of the extremities. In one instance trismus (lock-jaw) appeared in three-quarters of an hour. (*Orfila*, vol. 1, p. 449.) Such is the ordinary character of the symptoms in an *acute* case of arsenical poisoning, *i. e.*, where from a quarter to half an ounce of the poison has been taken. As a general rule, the symptoms in the acute form of poisoning which prove fatal are *continuous*. Sometimes, however, there are remissions and even intermissions, which may lead to a deceptive hope of recovery, or (by the recurrence of symptoms) to an erroneous supposition that a fresh quantity of poison has been administered. In the case of the Duc de Praslin, who died from the effects of a large dose of arsenic, the remissions in the symptoms during the week which he survived were such as to deceive the skilful physicians who attended him. At one time the vomiting had ceased, and at another time the pain;—the most persistent effects were the smallness and irregularity of the pulse and coldness of the limbs. (*Ann. d'Hyg.*, 1847, vol. 2, p. 391.) Dr. Maclagan met with two cases in which there were intermissions of a prominent symptom (vomiting) for one and three days respectively. The symptoms recurred without, so far as could be ascertained, any fresh dose of poison having been given to these persons. (*Ed. Monthly Jour.*, Jan. 1853.)

The whole of the symptoms here described may not be met with in every instance. Thus the *pain*, which is usually excruciating, described as a fire burning within the body, is sometimes absent. In a well-marked case of poisoning, which occurred in October, 1839, a dose of from one ounce to two ounces of arsenic was taken; there was no pain except of the most trifling character, just before death. It has been supposed that this symptom was frequently absent when the dose was large; but a case occurred in Guy's Hospital, in which only forty grains had been taken, and the patient died without complaining of pain. (*Guy's Hosp. Rep.*, vol. 4, p. 68.) There are many similar instances on record. Even when the stomach has been found intensely inflamed after death the patient had not complained of pain during the time which she survived. The symptoms of irritation of the stomach and bowels are seldom wanting, or there is vomiting, if there should be no purging. In one case of criminal poisoning with arsenic, in which I was consulted by Mr. Veasy, which was tried at the Bedford Spring Assizes in 1842, there was neither vomiting nor purging. A similar case is reported by Dr. Brown, of Lahore. A girl died in *three hours* after eating some sweetmeats poisoned with arsenic. There was neither vomiting nor purging. After death the stomach was found intensely inflamed, and it contained a large quantity of roughly powdered arsenic enveloped in mucus and an ounce of dark bloody fluid. The intestines were also inflamed. (*Medical Report of Bengal Presi-*

dency, 1869, p. 146.) The quantity of poison taken must have been small. In a case which occurred to Dr. Feital, although half an ounce of arsenic had been taken, there was no vomiting. (*Med. Times*, Dec. 12, 1846, p. 202.) Intense *thirst* is a common symptom, but even this is sometimes absent. With respect to the urinary secretion there is no certain rule; it is sometimes suppressed, as in several cases reported by M. Flandin; at other times it is natural, or only slightly diminished. (*Des Poisons*, vol. 1, p. 521.) It is necessary for a medical jurist to attend to these anomalies, as otherwise the symptoms of arsenical poisoning may be mistaken for those of disease.

The following cases are of interest, as showing the symptoms attending the acute form of poisoning, when arsenic has been taken in a small dose, insufficient to destroy life. I was consulted respecting these cases in December, 1857. At a large Industrial School near London, *three hundred and forty children* were suddenly seized with symptoms of poisoning by arsenic, soon after breakfast. They had been supplied with milk diluted with water from a boiler, into which a quantity of an alkaline solution of arsenic had been placed, under the notion that the alkaline arsenite would effectually cleanse it of fur. Two gallons of this *cleansing* liquid, containing about *nine pounds of arsenic*, perfectly dissolved by the aid of a large quantity of soda, had been well mixed with the water in the steam-boiler of the establishment, without any information being given of its dangerous properties! Fortunately only four gallons of the poisoned water were drawn out of the boiler. This quantity was mixed with thirty gallons of milk, and divided among the 340 children—about a gallon of the mixture being assigned to ten children. Upon an average each child took a *grain* of arsenic more or less. The nature of the poison was soon discovered, and proper remedies suggested and employed. It is remarkable that in this wholesale poisoning the symptoms varied but little among the children. There was shivering, with pain in the stomach and bowels, and in most of the cases vomiting of a clear, ropy, mucous fluid, of a green color (the cleansing liquid having this color). These symptoms were developed within one hour. In about three hours after the meal, pain in the forehead more or less intense, was a prominent symptom, and there was a copious discharge of a watery mucous fluid from the nose (coryza). Seven had cough of a croupy character, three vomited blood, and one passed blood by the bowels. Some suffered from inflammation of the stomach; of these six only were under treatment at the end of the first week, and one did not recover until after the second week. The treatment consisted in giving gum water with albumen, and in keeping up vomiting by emetics or warm greasy water for twelve hours; after this, castor oil was administered. The whole of the children recovered, and thus this occurrence did not become a subject of public investigation.

It might be supposed, *a priori*, that the symptoms of irritation occasioned by arsenic would be protracted in their appearance or mitigated in their character when the poison was taken mixed with opium; but in one well-marked case, in which a large dose of arsenic was swallowed with upwards of an ounce of laudanum, there was severe pain, abundant vomiting for two hours, and death took place in six hours. (*Med.-Chir. Rev.*, vol. 7, p. 170; also *Ann. d'Hyg.*, 1847, vol. 2; p. 199.)

It has been stated that stupor and other symptoms of cerebral disturbance were more likely to occur when the dose of arsenic was large; but a case was communicated to the *London Medical Review* (April, 1811, p. 188), by Mr. Soden, of Coventry, which shows that, with a large dose of arsenic and rapid death, there may be violent symptoms affecting the stomach and bowels, and few or none indicative of nervous disorder. A man, aged 22, purchased seven ounces of finely powdered arsenic, and swallowed, between seven and eight in the morning, not less than four and probably six ounces of the poison. In about half an hour he was found vomiting; there was severe pain in the abdomen, with a rapid pulse and slight convulsions of the legs. In two hours purging supervened, and there was constant inclination to pass urine; the pain in the bowels became almost intolerable, the convulsive motions of the limbs more frequent, and the pulse more feeble, but still very quick. He died in less than *four hours*, after a dreadful fit of convulsive laughter, his limbs becoming suddenly rigid (tetanus). In this case, there was neither stupor nor faintness, but there was severe pain, with convulsions. On inspection, the stomach was found highly inflamed, "the mucous coat looked as though it had been beautifully injected," and two ounces of arsenic were found in the cavity of this organ.

Chronic or Slow Poisoning.—Should the person recover from the first effects, and the case be protracted, or should the dose have been small and frequently administered, there will be inflammation of the conjunctivæ, with suffusion of the eyes, and intolerance of light—a condition which is, however, often present with the early symptoms above described. (*Med. Times*, Aug. 30, 1851, p. 229.) There is also great sensibility or irritation of the skin, accompanied by a vesicular eruption, which has been called "eczema arsenicale." Sometimes this has assumed the form of nettle-rash, or of the eruption attending scarlet fever, for which disease arsenical poisoning has been mistaken. Local paralysis, preceded by numbness, or tingling in the fingers or toes, and other symptoms of nervous disorder, are also very common consequences. Paralysis from arsenic is sometimes general, and affects both the upper and lower limbs. It may supervene on the cessation of symptoms of gastric irritation in cases of acute poisoning. It may be complete, or amount only to great weakness (*Annuaire de Thérapeutique*, 1858, p. 229). The patient becomes emaciated, and sinks exhausted. Exfoliation of the cuticle and skin of the tongue, with the falling off of the hair, has likewise been witnessed. (Case of the *Turners*, 1815, Marshall, pp. 14, 119. Husemann's *Jahresbericht*, 1871, p. 527.) *Salivation* has been observed to follow, especially when small doses of the poison have been given for a length of time. (*Med. Gaz.*, vol. 16, p. 790.) A well-marked case of this kind occurred to Mr. Jones, in which the effects produced by small doses of arsenic might have been mistaken for those of mercury. There was fetor of the breath, with superficial ulceration of the gums and throat. (*Med. Gaz.*, May 8, 1840, vol. 26, p. 266.) *Strangury* has also been noticed among the secondary symptoms. (Marshall, *On Arsenic*, pp. 44, 314.) From a statement of this author (*op. cit.*, p. 111), it appears that there was a yellow or jaundiced state of the countenance in one of the cases reported by him. A similar state of the countenance was noticed by Dr. Thomson in the case of L'Angelier—a fact which gave rise to some discussion at the trial of Madeline Smith. (*Irvine's Report*, p. 51.) The late Dr. Geoghegan observed, that in several cases of poisoning by arsenite of copper, jaundice was among the symptoms.

The following case of *slow* or *chronic poisoning* with arsenic is recorded by Flandin. It illustrates one form of secret murder, and is well calculated to inspire caution in trusting to symptoms as evidence of disease. A woman put daily into the soup of her fellow-servant a small quantity of white arsenic in powder. Shortly after dinner this person was seized with vomiting, which led to the rejection of the food and poison, before the latter had caused any serious mischief. As this practice was continued for about six weeks, the stomach became exceedingly irritable, there was pain in the bowels, and the woman became much emaciated. There was also spitting of blood, with such a degree of nervous irritability, that a current of air caused an attack of spasms and convulsions. When the patient found that she could not bear anything on her stomach, she left the place and passed two months in the country. Her health was gradually re-established there, and she returned to resume her usual occupations. The prisoner, however, renewed her attempts; and, to make sure of destroying life, gave her one morning in coffee, a strong dose of arsenic in powder; violent vomiting ensued, and the poison was expelled with the food taken at breakfast. Arsenic was detected in the vomited matter, and the explanation of the cause of the long previous illness then became clear. Under proper treatment the patient recovered. (*Op. cit.*, vol. 1, p. 510.) Such a train of symptoms might, from careless observation, be easily referred to chronic inflammation or ulceration of the stomach from natural causes, leading to perforation.

There are many anomalous cases on record in which the symptoms have diverged so much from the ordinary course as to embarrass medical practitioners. For some of these I must refer to a paper by Dr. Ogston (*Med. Gaz.*, vol. 47, p. 181, and Husemann's *Jahresbericht*, 1872, p. 481). A case of slow poisoning with arsenic may very easily be mistaken for gastro-enteritis, and treated accordingly. (See *Prov. Journ.*, Nov. 1843, p. 127, *Med. Times*, Aug. 21, 1874, and *Ann. d'Hygiène*, 1837, vol. 1, p. 347.) Dr. Pfeufer met with an instance of this masked form of arsenical poisoning. There was general and well-marked paralysis of the muscular system, and a complete loss of sensation in the fingers. It was only after a year that the patient began to recover the use of his limbs. It appeared that his wife had been in the habit of giving him small doses of arsenic in his food. None of the articles of food, or of the matters vomited, could be procured for analysis; nevertheless, the evidence of chronic poisoning from symptoms was considered to be sufficiently strong to justify a conviction. (*Zeitschrift für Rationelle Medizin*, B. vi, H. i, 102, Heidelberg, 1847.)

We have in these cases the usual characters of slow poisoning with arsenic, indicated by wasting fever and general derangement of the bodily functions. The effects of the Aqua Tofana, as used by mediæval poisoners, were very similar to these. Persons to whom this liquid was administered in small and frequent doses, died without the slightest suspicion of the cause of death being excited.

Arsenic not an Accumulative Poison.—It is well known that arsenic is carried into the circulation by absorption, and it is an important question regarding its medicinal use, whether its elimination in the living body takes place with the same rapidity as its absorption. I am not aware of any facts which show that arsenic can be taken in non-fatal (medicinal) doses for a certain period, accumulate in the body, and then suddenly give rise, without increase of dose, to all the marked symptoms of acute poisoning. On the contrary, all experience is in favor of the rapid elimination of this poison; and unless the doses are too frequently repeated, or too rapidly increased, no danger will accrue from the quantity administered. There are now numerous facts which show that, provided a sufficient interval is allowed to elapse between the doses, and each dose is not too large, this poison is rapidly thrown out of the system.

In treating a case of eczema in a boy, Dr. Wilks prescribed the twenty-fourth part of a grain of arsenic three times daily, making one-eighth of a grain per diem. This was continued for seventy days, so that in ten weeks the boy had taken nearly *nine grains* of arsenic, or a quantity sufficient to destroy four adults. I found by analysis that arsenic was daily eliminated in the urine; and after the medicine was discontinued, it still continued to pass away by this channel for about ten days, when the boy left the hospital. (See *ante*, p. 39.) Arsenic may remain in the body for a period of from sixteen to twenty-one days after the administration of it has ceased; but there is no evidence that it so accumulates in the system as to be suddenly productive of dangerous symptoms; and experience shows that if its use be discontinued, the viscera, after a few weeks, do not contain a trace of the poison. Hence the sudden occurrence of violent symptoms of vomiting and purging would, in general, justify the inference that another and larger dose of poison had been taken shortly before. Mr. Hunt considers that arsenic used medicinally does accumulate in the body. (*On the Skin*, 1847, p. 11.) The facts which he adduces, however, in support of this view, admit of another explanation. The speedy elimination by the kidneys proves that the poison is thrown out of the system; and where any morbid changes affect these or other eliminating channels, symptoms of poisoning may of course appear. Accumulation simply means, therefore, that the process of elimination is partially or entirely arrested. Of the latter condition no satisfactory proofs have yet been furnished.

CHAPTER XXXIII.

ARSENIC—APPEARANCES AFTER DEATH—CHANGES PRODUCED IN THE STOMACH—INFLAMMATION—ULCERATION—PERFORATION—GANGRENE—CHANGES IN THE INTESTINES—POISONING BY EXTERNAL APPLICATION—BY ABSORPTION—POISONING BY ARSENICAL FUMES—COPPER-SMOKE—QUANTITY OF ARSENIC REQUIRED TO DESTROY LIFE—FATAL DOSE—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

Appearances after Death.—The striking changes produced by arsenic are generally confined to the stomach and intestines. They are commonly well-marked in proportion to the largeness of the dose and the length of time which the individual has survived after taking the poison. Our attention must first be directed to the *stomach.* Arsenic seems to have a specific effect on this organ; for, however the poison may have entered into the system, whether through a wounded or ulcerated surface, or by the act of swallowing, the stomach has been found inflamed. Inflammation of this organ cannot, then, be always considered to depend on a local irritant action of the poison. The mucous membrane of the stomach, which is often covered with a thick layer of mucus, mixed with blood or bile, and with a thick white pasty-looking substance containing arsenic, is commonly found red and inflamed; the color, which is of a dull or brownish red, becomes brighter on exposure to the air; at other times it is of a deep crimson hue, interspersed with black-looking streaks or patches of altered blood between the rugæ. The redness is usually most strongly marked at the greater end; in one case it may be found spread over the whole mucous surface, giving to it the appearance of red velvet—in another it will be chiefly seen on the prominences of the folds (rugæ). It frequently assumes a dotted or striated form, stretching in curved lines between the two openings of the stomach. Blood of a dark color is effused in various parts between the folds, or beneath the lining membrane, an appearance which has been mistaken for gangrene. (See *ante*, p. 227.) The stomach usually contains a mucous liquid of a dark color tinged with blood. The coats are sometimes thickened in patches, being raised up into a sort of fungous-like tumor, with arsenic imbedded in them; at other times they have been found thinned; and in other cases of a gelatinous consistency and appearance. The mucous membrane is rarely ulcerated, and still more rarely gangrenous. Among numerous inspections I have not seen one instance in which gangrene of the coats of the stomach had resulted from the action of arsenic. In general, death takes place before gangrene is set up. In the case of the Duc de Praslin, who died from the effects of arsenic on the seventh day, a gangrenous state of the coats of the stomach is said to have been observed. Between the two apertures of the stomach there were seven large eschars—they were black, with yellowish-white margins, and of a different consistency from the adjoining coats. They were in some parts horny. Around these gangrenous spots, which did not involve the whole substance of the coats, the mucous membrane was somewhat softened and of a deep red color, evidently due to inflammation. (*Ann. d'Hyg.*, 1847, vol. 2, p. 396.) In one case a patch of effused blood was mistaken for gangrene, and the mistake led to a false charge of poisoning. (*The Queen* v. *Dore and Spry*, Aug. 28, 1848, and *Med. Gaz.*, Nov. 24, 1848.)

Perforation of the stomach, as a result of the action of arsenic, is so unusual an appearance, that there are but few accurately reported instances on record. In a case examined by M. Chevallier, the stomach of a person who had died from the effects of arsenic was found perforated at the larger end. The aperture is described to have been of the size of a franc-piece, round, soft, and somewhat thickened in its margin. There was no redness or sign of erosion about it, and there was no appearance of ulceration on other parts of the mucous coat. Externally the stomach was covered with false membranes, arising from inflammation of the peritoneum. (*Ann. d'Hyg.*, 1852, vol. 1, p. 448.) This case is so imperfectly reported that it is impossible to say whether the perforation was caused by arsenic, or whether it was the result of other morbid changes.

The mucous glands of the stomach have been found enlarged; but this is by no means an unusual morbid appearance from any cause of local irritation, without reference to poisoning. Various morbid appearances are said to have been met with in the lungs, heart, brain, and urinary organs; but they do not appear to be so characteristic of arsenical poisoning as to admit of a medico-legal use in enabling a medical man to distinguish poisoning from disease. It is chiefly to the stomach and intestines that a medical jurist must look for the basis of medical evidence in regard to the appearances after death. Dr. Wilks met with an ecchymosed condition of the lining membrane of the left ventricle of the heart in a case in which a man died in twelve hours from acute poisoning by arsenic. Dr. Greiner met with a fatty state of the liver and bloodlessness of the body in one case of acute poisoning with arsenic. (Horn's *Vierteljahrsschrift*, 1866, vol. 2, p. 345.) In one case, which I saw in June, 1857, in which a person died in twenty hours after he had taken two teaspoonfuls (300 grains) of arsenic, there was inflammation of the œsophagus as well as of the stomach, duodenum, and rectum. The mucous membrane had a reddish-brown color. At the pyloric end of the stomach, as well as in the duodenum, there were several black patches from effused and altered blood. There was congestion of the brain and its membranes, with enlargement of the liver; but these appearances were not referable to the action of arsenic. A small quantity of the poison was found in the thick reddish fluid found in the stomach; but the greater part of the large dose taken had been thrown off by vomiting.

A witness may be asked how long a time is required after the taking of the poison, for the production of these well-marked appearances in the stomach, more especially of *inflammation* of the mucous membrane. In reference to this question, there are the following facts. In a case which I was required to examine, a large dose of arsenic had been taken;

the man, aged 21, died in *five hours*, and the stomach was found intensely inflamed, especially about the greater curvature. In a case that occurred to Mr. Thompson, of Nottingham, half an ounce of the poison was taken; the patient died in *six hours*, and the stomach was found uniformly red and inflamed. In another that occurred to Dr. Booth, of Birmingham, the same quantity of arsenic was taken; the patient died in *six hours and a half;* on inspection, the gullet was inflamed, the whole internal surface of the stomach was of an intense scarlet color, and there was redness and increased vascularity of the small intestines. In three cases of poisoning with arsenic which occurred to Mr. Foster, of Huntingdon, death took place in one, a child, at the end of *two hours;* in the second, an adult, at the end of *three hours and a half;* and in the third, after the lapse of about six hours. In each of these the stomach was found highly inflamed, and in the one that proved fatal in two hours, the mucous membrane had a vermilion hue. Mr. Clegg, of Boston, communicated to me a case in which a woman died within two hours after taking a large dose of arsenic. On inspection, the whole of the mucous membrane of the stomach was found intensely inflamed, and upwards of an ounce of solid arsenic was spread over it in a pasty state.

Another question put to a witness may be this: What period of time is required for *ulceration* of the mucous membrane to take place, as an effect of this poison? If arsenic has destroyed life with unusual rapidity, and the stomach is found ulcerated, an attempt may be made to refer this ulceration to some other cause. Such an attempt was made in the case of Rhymes, which was the subject of a criminal trial at Reading, in 1841. (*Guy's Hospital Reports*, Oct. 1841, p. 283.) I found ulceration of the mucous membrane, which had been completely removed in patches, although the deceased had survived the effects of the poison only *ten* hours. The deposition of the arsenic in and around the ulcers, as well as the appearance of recent inflammation about them, left no doubt that they had been produced by the poison, and were not owing to previous disease, as it was attempted to be urged in defence. When no arsenic is found in the stomach, a defence of this kind will carry with it considerable plausibility. In Waring's case a medical witness was questioned upon this point. The deceased was stated to have died from the effects of arsenic in *four hours;* the coats of the stomach were found ulcerated, but no poison could be detected in the organ. The witness admitted, on cross-examination, that it was contrary to experience that ulceration should be occasioned by an irritant poison in so short a time as four hours; but he nevertheless contended that this was the true cause. On such points, we can only be guided by observation; and one well-observed case is sufficient to place the possibility of ulceration being produced by arsenic, within a few hours, beyond all question. Sir R. Christison mentions a case observed by Mr. Hewson, where many eroded spots existed on the stomach, although the person had died from the effects of arsenic in *five* hours. (*On Poisons*, p. 340.)

But are the stomach and intestines always found inflamed in cases of poisoning with arsenic? The answer must be decidedly in the negative. At the trial of McCracken, at the Derby Autumn Assizes, in 1832, for killing his wife with arsenic, the fact of poisoning was clearly established, and a large quantity of arsenic was found in the stomach of the deceased; but there was no appearance of inflammation, either in this organ or the intestines. The two following cases are recorded in *Rust's Magazine.* A servant girl had some arsenic administered to her in chocolate. She was seized with nausea and violent pain in the stomach, and died the same evening. On inspection, there was no remarkable redness or inflammation of the stomach; but arsenic was found in the duodenum. A man was taken ill with vomiting and violent pain in the abdomen after partaking of some soup, and he died from symptoms of poisoning. On inspection, the mucous surface of the stomach presented no morbid change, with the exception of slight redness about the cardia. Arsenic was found in the contents of the intestines. In a case quoted by Flandin from Etmuller, a girl swallowed a large dose of arsenic, and died twelve hours afterwards, without having vomited, or manifested any symptoms. On inspection, arsenic was found in the stomach, but there was no material change in the organ. (*Op. cit.*, vol. 1, p. 234.) In a case tried at the Hertford Lent Assizes, 1855 (*Reg.* v. *Newton*), the coats of the stomach were thickened and pulpy, but were entirely free from inflammation. Death was clearly caused by arsenic. In another case, the mucous membrane of the stomach was found so pale that, at first, death from poisoning by arsenic was not suspected. Even with symptoms of irritation of the stomach, well-marked appearances may be wanting. Occasionally the appearances are so slight, that were not the attention of the examiner especially directed to the fact of poisoning, they would be passed over. (See case by Dr. May, *Prov. Med. Jour.*, July 16, 1845, p. 453.) These exceptional cases appear to show that arsenic does not exert any local action of a chemical nature, like a corrosive, on the stomach; for the action of corrosives takes place on mere contact, without reference to the state of constitution, or the quantity of poison taken. Medical evidence of poisoning from appearances after death is in such cases entirely wanting; they are not very common, but still their occurrence proves that unless great care be taken in forming an opinion, a case of arsenical poisoning may be overlooked. They teach this important fact in legal medicine, that the non-existence of striking changes in the alimentary canal after death, is no proof that the party has not died from the effects of irritant poison. When the dose of arsenic is small, well-marked changes in the body are rarely met with.

In some instances the mouth, pharynx, and *gullet* have been found inflamed, but in general there are no appearances in this part of the alimentary canal to attract particular attention. The mucous membrane of the *small intestines* may be inflamed throughout, but commonly the inflammatory redness is confined to the *duodenum*, especially to that part which joins the pylorus. Of the large intestines, the *rectum* appears to be the most prone to inflammation. The liver, spleen, and kidneys present no changes which can be considered characteristic of arsenical poisoning, although these, like the other soft organs of the body, are receptacles of the absorbed poison. It is worthy of remark, in relation to the known antiseptic properties of arsenic, that the parts specially affected by this poison (the stomach and intestines) occasionally present the well-marked characters of irritant poisoning for a long time after death. This was established in the case of the *Queen* v. *Dazley*, tried at the Bedford Summer Assizes, July, 1843. The prisoner was convicted of poisoning her husband with arsenic, upon evidence obtained by the exhumation and examination of the body six months after interment. The stomach and intestines were the only parts of the body undecomposed. This case presents many important subjects for reflection to the medical jurist; as, for example, the substitution of arsenic for medicine—the length of time after death at which good evidence may be obtained from the dead body—the fact of another person laboring under symptoms of poisoning with arsenic, who had accidentally partaken of the supposed medicine—and, lastly, the evidence from the death of an animal which had swallowed some of the matter vomited by the deceased. In two cases (*Reg.* v. *Chesham*) referred to me by Mr. Lewis, coroner for Essex, a deep red inflammatory appearance of the mucous membrane, immediately below a layer of sulphuret of arsenic, was well marked, although the bodies had been buried *nineteen* months. In a case which occurred in March, 1848, the stomach was also well preserved; and it retained an inflammatory redness after the lapse of *twelve* months. Absorbed arsenic does not appear to prevent the decomposition of the soft organs in which it is deposited. For a summary of the appearances caused by arsenic, and its influence in modifying putrefactive changes, I must refer the reader to a paper by the late Dr. Geoghegan in the *Medical Gazette*, vol. 46, pp. 178 and 218, and "Observations on Arsenical Poisoning," *Dublin Quarterly Journal*, Feb. 1851.

Poisoning by External Application.—The effects produced by arsenic when applied to the unbroken or diseased skin or to wounds have been elsewhere fully considered, *ante*, p. 26. The following case may serve as a type of this form of poisoning, which is not very common. In February, 1864, I was required to investigate a case of suspicious death which occurred near Halesworth, in Suffolk. A girl, æt. 9, died rather suddenly, after an illness of about ten days. The mother had rubbed some white precipitate ointment mixed with arsenic on the head of the child, which was diseased. Her object, she stated, was to kill the vermin on the scalp. No symptoms of note were observed until about the fifth day after the application, when the child appeared ill and complained of thirst. On the eighth day she was very unwell; there had been cramp, with slight action on the bowels, but no vomiting. She became drowsy, and died on the tenth day. Mr. Haward examined the body, and forwarded to me the viscera for chemical analysis, the case being very obscure. The lining membrane of the stomach and duodenum was inflamed; in the stomach the inflammation was well marked towards the greater end: these were the principal post-mortem appearances. Traces of arsenic were found in the mucous fluids of the stomach, in the coats of the stomach and intestines, and in four ounces of the liver, but arsenic in a *solid* form could nowhere be detected. A portion of the diseased hairy scalp was examined, and yielded arsenic as well as mercury (from white precipitate) in large proportion, the arsenic being estimated at from two to three grains. From the evidence given at the inquest there was no doubt that the mother's account was correct, and that her child had died from the ignorant application of arsenic externally to a diseased portion of the scalp. (*Guy's Hosp. Rep.*, 1864, p. 220.)

The remarkable features of the case were these: No symptoms appeared until after the *fourth day*, and then only great thirst; there was slight purging with cramps on the eighth day, and death took place on the tenth, without any vomiting. Arsenic was found in the stomach and contents, and its presence there might have led to an erroneous inference of its having been criminally administered by the mouth. It was, however, merely in traces, and obviously enough the result of mucous elimination. The nature and mode of occurrence of the symptoms were opposed to any other presumption. That absorbed arsenic may be thus transferred from the blood to the stomach and intestines, has been distinctly proved by the experiments of Dr. Pavy and myself. (*Guy's Hospital Reports*, 1860, vol. 6, p. 397; also *ante*, p. 56.)

Arsenic in Vapor—Arsenical Fumes—Copper Smoke.—It is not often that a case is heard of in which white arsenic has caused death by reason of its having been breathed or swallowed in the state of vapor. In April, 1858, a case involving the effects of arsenical vapors was the subject of an inquest in London (see ON POISONS, 2d edit., p. 234); but on that occasion there was an entire failure of proof that the arsenical vapor was the cause of death. I am indebted to Mr. Oxley, of Rotherham, for the account of a case which fell under his notice, which was the subject of a trial at the York Lent Assizes, 1864.

The prisoner placed some burning pyrites containing arsenic at the entrance of the door of a small room in which there were eight children, including an infant in a cradle. From the evidence, it appeared that all the children suffered from the fumes, which were chiefly those of sulphurous acid. A canary that was in a cage died from the effects. The children were speedily removed from the house, and recovered, but the infant was left there for an hour. It suffered from vomiting, and when seen by Mr. Oxley, about seventeen hours afterwards, the child was pulseless. It vomited incessantly, was much purged, and appeared to be in great pain. It died about twenty-four hours after exposure to the fumes. On inspection, the stomach and intestines were slightly inflamed. The brain and lungs were congested, and the lining membrane of the trachea was of a bright red color. Dr. Allan detected arsenic in the contents of the stomach, in the lungs, in the coats of the stomach, and the spleen. None was found in the liver. The pyrites contained arsenic, and gave off while burning arsenious acid in vapor and sulphurous acid. Some of the appearances were owing to sulphurous acid, but death was probably caused by arsenic. The prisoner was found guilty of manslaughter. A case is reported in which it is stated that the members of a family were made ill by arsenical vapors arising from the walls of a room, and that one of them died. (*Pharm. Jour.*, July, 1870, p. 66.)

The vapors which escape from the arsenic and copper-smelting works of Cornwall and South Wales are those of arsenious acid, or white arsenic. The arsenic issues from the flues as a thick white smoke; and when no precautions have been taken, it has destroyed cattle as well as vegetation to a great extent. In the grinding mills it may be respired as a fine dust. Great precautions are taken by the workmen, by plugging the nostrils and covering the mouth, to avoid breathing this arsenical dust, but in spite of these accidents do occur. The men who remove the impure arsenic from the flues suffer from severe pustular and scaly eruptions, affecting the scrotum and other parts of the skin where there are hollows or depressions. Thus, according to Dr. Jago, the parts especially liable to be attacked, next to the scrotum, are the depressions between the lower lip and chin, the angles of the nose and face, and the lines along the forehead; in fact, every crevice in which the arsenical dust can lodge. This appears to indicate a local irritant action. Among the constitutional effects there have been noticed: cough, with shortness and difficulty of breathing, debility, emaciation, profuse perspiration on slight exertion, scantiness of urine, and increased pulsations of the heart. The tongue presents red edges, with a white fur, and the gums are inflamed. Frequent nausea and vomiting are also among the symptoms. (See paper by Mr. Kesteven, *Assoc. Med. Jour.*, Sept. 1856, p. 811.) A few years since an inquest was held on the body of a child at Plymouth, whose death was erroneously referred to arsenical fumes escaping from burning minerals. In Cornwall, the deaths of workmen may sometimes be traced to the poisonous vapor; but, on the whole, considering the nature of their occupation, the men enjoy average health. Many cannot work long in the arsenic factories, while others have continued to work in them for twenty or thirty years.

In the case of the *Queen* v. *Garland* (Cornwall Lent Assizes, 1851), a prosecution for nuisance and damage from arsenic works, it was proved that animal and vegetable life suffered, to a great extent, from these fumes. Horses and cattle perished, and, before death, they became much emaciated, and lost their hair. It would appear that these effects are produced as much by the animals pasturing on poisoned herbage as by the actual breathing of the arsenical vapors. After death, the stomach and bowels were found inflamed, and sometimes mortified. Donkeys were especially liable to suffer, owing to these animals eating thistles and plants with hard and irregular surfaces, favorable for the retention of the arsenical dust. In South Wales the animals within the range of the arsenical smoke from the copper works have, in addition to other symptoms, suffered from enlargement and diseases of the joints. These facts, it will be perceived, are quite adverse to the Styrian hypothesis of the fattening effects of arsenic on horses and cattle.

Quantity required to destroy Life—Fatal Dose.—Arsenic has been safely given in medicinal doses of from 1-20th to 1-8th of a grain; but when the dose is raised to half a grain, or a grain is given at once, symptoms of poisoning show themselves. Adults and even children have, however, recovered from larger doses accidentally administered, as the following cases will show: At a large dinner-party, in London, in October, 1839, it was observed that three persons, who had partaken of the port wine on the table, were seized with symptoms of poisoning. The wine was suspected to contain poison, and it was sent to me for examination. It was clear, of the usual color, and odor, and possessed all the characters of good wine; but there was a small quantity of a reddish-white crystalline sediment at the bottom of the bottle. From the account of the symptoms the wine was suspected to contain arsenic. This was found to be the case, and the quantity of poison dissolved amounted to about 1.2 grain in each fluid ounce. The following were the facts: A child of about sixteen months took a quantity of the wine, containing about *one-third of a grain* of arsenic. In twenty minutes this child became sick, vomited violently for three hours, and then recovered. A lady, aged 52, took a quantity of wine containing rather less than *two grains* of arsenic. In about half an hour she experienced faintness. Violent vomiting came on, and lasted four hours, but there was no pain. She then gradually recovered. A gentleman, aged 40, took a quantity of the wine, containing rather more than *two grains* of the poison. The symptoms in him were similar, but more severe; and had he taken another glass of the wine, it is probable that he would have been killed. It may be proper to observe that although this wine was perfectly saturated with arsenic, not the least taste was perceived by any of the parties. The smallest fatal dose hitherto recorded was observed in a case communicated by Dr. Castle, of Leeds, to the *Provincial Journal* (June 28, 1848, p. 347). A woman took half an ounce of Fowler's solution (arsenite of potash) in unknown doses, during a period of five days. She then died, and on examination the stomach and intestines were found inflamed. Death took place by syncope (mortal fainting), and there was an absence of vomiting and purging. The quantity of arsenic which here destroyed life could not have been more than *two grains*. In another case, two grains and a half of arsenic, contained in two ounces of fly-water, killed a robust, healthy girl, aged 19, in thirty-six hours. (See *Med. Gaz.*, vol. 39, p. 116.) Hence, under circumstances favorable to the operation of the poison, the fatal dose in an adult may be assigned at from *two to three grains.*

Dr. Lachèse, who has examined this question, states that from one to two grains may act fatally in a few days. (*Ann. d'Hyg.*, 1873, vol. 1, p. 334.) It is probable that this dose would prove fatal to a child, or to weak and debilitated persons.

The cases of the children of the Industrial School at Norwood (p. 291), show that, except under peculiar circumstances, a dose of one grain is not likely to prove fatal.

There are some instances of recovery from large doses, varying from sixty grains (*Med. Gaz.*, vol. 2, p. 771, and vol. 19, p. 258) to two ounces (*Lancet*, Oct. 2, 1852, p. 299, also 1857, vol. 2, p. 114); but these are exceptions to the rule. In these cases the arsenic has been commonly taken on a full stomach and rapidly ejected by vomiting.

Period at which Death takes place.—Large doses of arsenic commonly prove fatal in from eighteen hours to three days. The average time at which death takes place is twenty-four hours; but the poison may destroy life within a much shorter period. There are now many authentic cases reported, in which death has occurred in from three to six hours. In 1845 I met with a well-marked case of death from arsenic in five hours; and in another, which occurred in April, 1849, death took place in two and a half hours. (*Guy's Hosp. Rep.*, Oct. 1850, p. 183. See also *Ann. d'Hyg.*, 1837, vol. 1, p. 339.) Mr. Foster, of Huntingdon, met with the case of a child under three years of age, who died within *two hours* from the effects of arsenic. The quantity taken could not be determined. A case also fatal in two hours occurred to Mr. Clegg (p. 297). In some of these cases of rapid death, the brain and nervous system have been observed to be affected, the patient suffering from narcotism and convulsions; but this by no means implies that symptoms of irritation are absent. In Mr. Soden's case (p. 292), in which not less than four, and probably six, ounces of the poison had been taken, the patient died in less than *four hours*, and two ounces of arsenic were found in the stomach. We have here an instance, which occurred in March, 1810, of arsenic destroying life and producing excessive inflammation in less than *four hours;* and yet at a criminal trial, sixteen years afterwards (Lewes Assizes, 1826, case of Russell) it was a debated question with some of the medical witnesses, whether it was possible for a person to die from the effects of arsenic in less then *seven* hours, and respectable medical authorities were actually quoted against this view! The following rapidly fatal case was communicated to me by Mr. Thompson. It is that of a youth, æt. 17, who died in April, 1860, from the effects of a large dose of arsenic, the symptoms from which he suffered being of a tetanic character. The poisoning was the result of an accident at Ramsey, in the Isle of Man. The medical evidence at the inquest was to the effect that not more than *twenty minutes* had elapsed between the time at which deceased sat down to eat his supper, containing the poison, and his death; but it is doubtful whether the poison may not have been taken some time before the meal.

In some instances death does not occur until long after the average period. In one case, in which an adult swallowed about half an ounce, death did not take place for *fifty hours*, and it is remarkable that there was an entire absence of pain. (*Med. Gaz.*, vol. 48, p. 446.) In the case of the Duc de Praslin, one large dose was taken, but death did not occur until the *sixth* day. (*Ann. d'Hyg.*, 1847, vol. 2, p. 367.) In October, 1847, a man who had swallowed 220 grains of arsenic was admitted into Guy's Hospital. He died on the *seventh* day. It is obvious that a patient who recovers from the first effects of this poison may still die from exhaustion or other secondary causes many days or weeks after having taken it, even although the whole of the poison has been eliminated from the body. Thus in the case of Dr. Alexander, death took place on the *sixteenth* day; and although a large quantity had been taken, no arsenic was found in the body. (*Med. Times and Gaz.*, April 18, 1857, p. 389.) In one instance in which arsenic was applied externally to the head, the person did not die until the *twentieth day*. The longest duration of a case of poisoning by arsenic is probably that reported by Belloc. A woman, æt. 56, employed a solution of arsenic in water to cure the itch, which had resisted the usual remedies. The skin became covered with an erysipelatous eruption, and the itch was cured, but she experienced severe suffering. Her health gradually failed, and she died after the lapse of *two years*, having suffered during the whole of this period from a general tremor of the limbs. (*Cours de Méd. Lég.*, p. 121.)

Treatment.—If vomiting does not already exist as a direct effect of the poison, emetics should be given and the effects promoted by mucilaginous drinks, such as linseed tea, milk, or albuminous liquids. When none of these can be procured, powdered mustard, in a proportion of from one to two teaspoonfuls in a glass of warm water, or failing this, a glass of hot greasy water, which may be procured in every household, should be administered at intervals. A saponaceous liquid, made of equal parts of oil and lime-water, has been given with benefit. While the oil invests the poison, the lime tends to render less soluble that portion of arsenic which is dissolved. The stomach-pump may be usefully employed; but unless the patient is seen early, remedial means are seldom attended with success. It is proper to examine occasionally by Reinsch's process (see p. 313) the liquid vomited, or withdrawn by the stomach-pump. We may thus ascertain when the poison is entirely removed from the stomach. In the event of the arsenic disappearing from the stomach and the person recovering from the first effects, it will be necessary to examine the urine which is passed daily. Arsenic may be found in this secretion for two or three weeks, or longer. Four ounces of urine are commonly sufficient for this observation. It should be concentrated by evaporation, and examined by Reinsch's process.

I have known death to occur in a case in which it was found, on subsequent examination, that every particle of poison had been removed from the stomach; and there are many instances of recovery on record, in which the arsenic appears to have been early ejected by constant vomiting and purging, or in which the free use of castor-oil with milk and barley-water has proved efficacious. The reader will find elsewhere reported the cases of three children, who took a quantity of white arsenic, and who perfectly recovered under this treatment, even where the circumstances were adverse to recovery. Had any of the so-called antidotes been given, they would have been credited with the recovery, whereas the real cause was the removal of the poison from the stomach and bowels by vomiting and purging. (*Guy's Hosp. Rep.*, 1865, p. 280.)

When arsenic has been taken in the form of powder, no confidence can be placed in the use of the so-called chemical antidotes—*hydrated sesquioxide of iron* or *hydrate of magnesia*. These substances may serve mechanically to suspend the poison, and thus facilitate its ejection from the stomach; but in this respect they possess no advantages over albumen or other viscid liquids. When arsenic has been taken in solution, the hydrated oxide of iron, if given in large quantity, will precipitate the poison in an insoluble form, and it may then become beneficial. A mixture of hydrate of magnesia and persulphate of iron may be used. The two chemical antidotes, as they are called, are present in the mixture, and the sulphate of magnesia produced by decomposition tends to act on the bowels and expel the poison.

The oxide of iron appears to have no more effect on *solid* arsenic than so much powdered brickdust, and to rest upon this as a neutralizer of the poisonous action of solid arsenic would be a delusion. Dr. Geoghegan noticed, in examining the stomach of a man who had died from arsenic, and to whom this substance had been freely given, that the white particles of arsenic were lying unchanged in the midst of the oxide. Casper made the same observation in the case of a youth who died from a dose of arsenic in twenty-four hours. About a *pound* of the oxide was found in the stomach after death, and beneath this, the white particles of arsenic could be seen by a lens lying upon the surface of the mucous membrane. (*Handbuch der Ger. Medicin*, 1857, vol. 1, p. 419.)

Our treatment must be directed to the early and entire removal of the poison from the stomach and bowels by emetics and quickly acting purgatives, such as castor-oil. Orfila has recommended that diuretics should be employed, in order to promote the secretion of urine, and thus favor the more speedy elimination of the poison from the system. If the secretion is suppressed, it would be desirable to restore it.

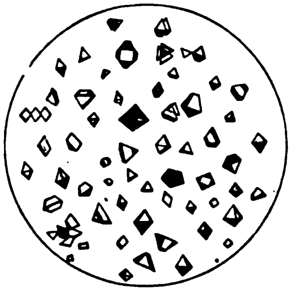
CHAPTER XXXIV.

ARSENIC—CHEMICAL ANALYSIS—TESTS FOR ARSENIOUS ACID—REDUCTION PROCESS—PROPERTIES OF ARSENICAL AS CONTRASTED WITH OTHER SUBLIMATES—SILVER AND COPPER TESTS—SULPHUR TEST AND PROPERTIES OF THE SULPHIDE OF ARSENIC—THE HYDROGEN TEST—MARSH'S PROCESS—REINSCH'S PROCESS—RESULTS OF THE TWO PROCESSES COMPARED—EVIDENCE FROM SMALL QUANTITIES OF ARSENIC.

ARSENIC—CHEMICAL ANALYSIS.

Arsenic as a Solid.—In the *simple state*, white arsenic may be identified by the following properties: 1. A small quantity of the powder, placed on platinum-foil, is entirely volatilized at and below a heat of 370° in a white vapor. Should there be any residue, it is impurity. Sometimes plaster of Paris or chalk is found mixed with it. If a small portion of the white powder is gently heated in a glass tube of narrow bore, it will be sublimed and form a ring of minute octahedral crystals, remarkable for their lustre and brilliancy. (See Fig. 15, also Fig. 17, p. 306.) Under a microscope of good magnifying power (250 diameters), the appearance of these crystals is remarkably beautiful and characteristic; one not exceeding the 4000th of an inch in size may be easily recognized by the aid of this instrument.

FIG. 15.



Crystals of arsenious acid by sublimation, magnified 30 diameters.

The form is that of the regular octahedron, of which the sides are equal. The crystals are frequently grouped, or nucleated; the solid angles are sometimes cut off, and occasionally equilateral triangular plates are seen. The forms are various; but all are traceable to the octahedron. Crystals which do not exceed the 10,000th, or even the 16,000th of an inch in diameter, present these microscopical characters distinctly; and the 1000th part of a grain of white arsenic will furnish many hundreds of crystals visible under the microscope. Generally speaking, the smallest crystals are those in which the octahedral form is the best defined. 2. If a portion of the powder be introduced on a fine platinum-wire into the edge of a smokeless flame, it will impart a steel-blue color, and evolve a white vapor. It will be observed, in these experiments, that white arsenic in vapor possesses no odor. 3. On boiling a small quantity of the powder in distilled water, it is not readily dissolved; but it partly floats in a sort of white film, and is partly aggregated in small white masses on the surface of the liquid or at the bottom of the vessel. It requires long boiling, in order that it should be dissolved and equally diffused through water (p. 287, *ante*). The floating of arsenic in fine powder takes place whether the water is hot or cold, and whether it is added to the poison or the poison to the water. This property of arsenic has given rise to some important questions on criminal trials. In *Reg.* v. *Lever* (Central Criminal Court, June, 1844) a question arose whether arsenic would float on tea. (See also the case of *Reg.* v. *Smith*, Wells Lent Assizes, 1869.) I have observed that the film formed on putting powdered arsenic into a vessel of cold water remained for five weeks on the surface, notwithstanding the occasional agitation of the vessel. 4. On adding a few drops of a solution of potash to the mixture of arsenic and water, and applying heat, the poison is entirely dissolved, forming a clear solution of arsenite of potash. 5. The powder is soluble by heat in strong hydrochloric acid, and when a piece of bright copper is immersed in the solution, it acquires a dark iron-gray stain from the deposit of metallic arsenic. If a portion of this solution is added to a strong solution of chloride of tin and boiled, a brownish-black deposit of metallic arsenic takes place. (Bettendorff's test.) 6. When the white powder is treated with a solution of sulphide of ammonium, there is no immediate change of color, as there is with most metallic poisons. On heating the mixture, the white powder is dissolved; and on continuing the heat until the ammonia is expelled, a

FIG. 16.

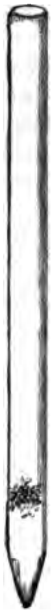
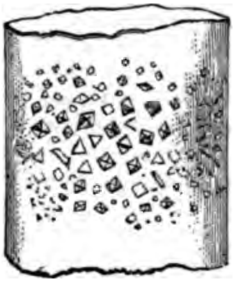


FIG. 17.



16. Small reduction-tube, with a crystalline sublimate of arsenious acid.

17. The tube, with sublimate magnified, showing the octahedral form of the crystals. These are shown still more highly magnified at p. 308.

rich yellow or orange-red film is left (sulphide of arsenic), which is soluble in all alkalies, and insoluble in hydrochloric acid. This yellow compound is produced from the mixture by spontaneous evaporation. A solution of sulphuretted hydrogen colors white arsenic slowly, and leaves by evaporation the same yellow compound. 7. It is oxidized and dissolved when heated in strong nitric acid; and on evaporation to dryness on a sand-bath, it leaves a white deliquescent residue (arsenic acid), which, when dissolved in a few drops of water, produces a brick-red colored precipitate with a solution of nitrate of silver. 8. When the powder is heated in a tube with two or three parts of charcoal, or any carbonaceous flux, it yields an iron-gray sublimate of metallic arsenic, which has an odor of garlic as it is evolved in vapor. This is called the "reduction test or process."

Reduction Process.—When a small portion of the powder, *i. e.*, from one-fourth to one-twentieth part of a grain, is heated with some reducing agent containing carbon, such as *soda flux* (obtained by incinerating acetate or tartrate of soda in a close vessel), in a glass tube about three inches long and from one-eighth to a quarter of an inch in diameter, it is decomposed; a ring of metallic arsenic, of an iron-gray color, is sublimed and deposited in a cool part of the tube. A mixture of one part of cyanide of potassium with three parts of dry (anhydrous) carbonate of soda forms an excellent flux for the reduction of arsenic. The materials and tube should be well dried. About two or three parts of either flux to one part of arsenic will be found sufficient. Any visible quantity of arsenic will serve for the purpose. In the absence of these fluxes, powdered ferrocyanide of potassium may be used in a similar proportion. After heating, a minute trace of arsenic remains in the flux; this cannot be expelled by heat. During the reduction, there is a perceptible odor, resembling that of garlic, which is possessed by metallic arsenic only, while passing from the state of vapor into arsenious acid. This *odor* was at one time looked upon as peculiar to arsenic, but no reliance is now placed on it as a matter of medical evidence —it is a mere accessory result. In this experiment of reduction, there are frequently two rings deposited in the tube (Fig. 19); the upper and larger ring has a dark-brown color, and appears to be a mixture of finely divided metallic arsenic and arsenious acid; the lower ring is small, of an iron-gray color, and consists of the pure metal.

FIG. 18.

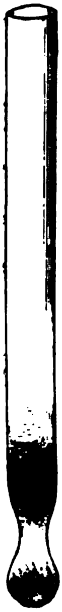
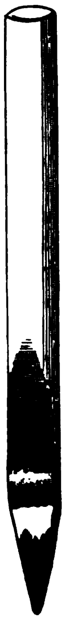


FIG. 19.



18. Berzelius's reduction-tube, with sublimate of metallic arsenic.

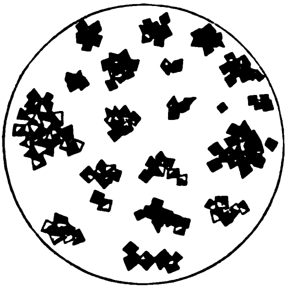
19. Ordinary reduction-tube, with two sublimates; the upper, brownish black; the lower, the pure metal in an annular deposit.

In order to determine the *weight of a sublimate*, the glass tube should be filed off closely on each side of the metallic ring, and weighed; the sublimate may then be driven off by heat, and the piece of glass again weighed; the difference or loss represents the weight. These sublimates are remarkably light, and require to be weighed in a delicate balance. An apparently large one weighed only .08 grains.

By heating gently the tube containing the sublimate (reduced to powder) in a dry test-tube, the metallic arsenic, during volatilization, forms octahedral crystals of arsenious acid, which, after examination by the microscope, may be dissolved in a few drops of water, and tested by one or more of the liquid reagents. The metallic sublimate, or the crystals produced from it, may be further subjected to the following process: Break the glass on which the sublimate is deposited, into fragments, and digest these in a few drops of the strongest fuming nitric acid, containing nitrous acid previously proved to be free from arsenic. The sublimate is thereby converted into *arsenic acid*. The acid solution should be evaporated to dryness; the white uncrystalline residue obtained should be dissolved in a few drops of distilled water, and a strong solution of nitrate, or of ammonio-nitrate of silver, added in small quantity to the residue. A brick-red coloration indicates arsenic acid, and thus proves incontestably that the sublimate was of an arsenical nature.

The upper or brownish-looking sublimate (Fig. 19), may be readily converted into one of the pure metal, by gently warming it in the flame of a spirit-lamp. Arsenious acid is then volatilized, and an iron-gray deposit of metallic arsenic appears. If the heat is continued, the whole of the metallic sublimate is volatilized and deposited in a cool part of the tube, in transparent and colorless octahedra of arsenious acid. This is the special character of an arsenical sublimate; it may be thus distinguished from sublimates of all metals or metalloids. The lower metallic sublimate procured by reduction (Fig. 19) may appear not in an annular form, but in detached nucleated particles of a somewhat globular shape. These are of an iron-gray color, quite unlike sublimed mercury, and when examined by the microscope, it may be seen that they consist of crystalline masses, and that they are angular, and not strictly spherical. This sublimate is sometimes produced in the last stage when the residue in the tube is strongly heated, and the air is exhausted.

FIG. 20.



Crystals of arsenious acid from a solution in water, magnified 124 diameters.

The *process of reduction* with the corroborative results above mentioned, is, when thus applied, conclusive of the arsenical nature of the substance under examination. Cadmium, selenium, and mercury produce sublimates, but these do not possess the appearance or properties of an arsenical sublimate.

Arsenic in Solution in Water—Liquid Tests.—A solution of arsenious acid is clear, colorless, possesses scarcely any perceptible taste, and has but a feebly acid reaction. In this state, we should first evaporate slowly a few drops on a glass-slide, when a crystalline residue will be obtained. On examining this with a microscope, it will be found to consist of numerous minute octahedral crystals, presenting triangular surfaces by reflected light. (See illustration, Fig. 20.)

1. *Silver Test.*—On adding to the solution *ammonio-nitrate of silver*, a pale yellow precipitate of arsenite of silver falls down; changing, under exposure to daylight, to a greenish-brown color. The test is made by adding to a strong solution of nitrate of silver, a weak solution of ammonia, and continuing to add the latter, until the brown oxide of silver, at first thrown down, is almost redissolved. The yellow precipitate is soluble in nitric, tartaric, citric, and acetic acids, as well as in ammonia.

2. *Copper Test.*—On adding to a solution of arsenic *ammonio-sulphate of copper*, a light green precipitate (arsenite of copper) is formed, the tint of which varies according to the proportion of arsenic present, and the quantity of the test added; hence, if the arsenic is in small proportion, no green precipitate at first appears; the liquid simply acquires a blue color from the test. In less than an hour, if arsenic is present, a bright green deposit is formed, which may be easily separated from the blue liquid by decantation. This test is made by adding ammonia to a weak solution of sulphate of copper, until the bluish-white precipitate, at first produced, is nearly redissolved; it should not be used in large quantity if concentrated, as the deep blue color tends to obscure or conceal the green precipitate formed. The precipitated arsenite of copper is soluble in all acids, mineral and vegetable, and in ammonia, but not in potash or soda. If a small quantity of the blue ammoniacal solution of this precipitate is poured over a crystal of nitrate of silver, a film of yellow arsenite of silver will appear around the crystal, by the production of arsenite of silver. If a strong solution of nitrate is added to the blue liquid, nearly neutralized by diluted sulphuric acid, a yellow precipitate of arsenite of silver is also produced. Thus the silver and copper tests may be employed with one quantity of liquid. The *dried* precipitate of arsenite of copper, when slowly and moderately heated in a well-dried reduction-tube, yields a ring of octahedral crystals of arsenious acid—oxide of copper being left as a residue.

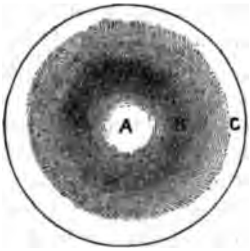
No chemist in the present day would think of employing these liquid tests in solutions, in which arsenic was mixed with *organic matter*. Almost all liquids used as articles of food are precipitated or colored by one or both of them, somewhat like a solution of arsenic, although none of this poison is present. Thus, then, any evidence founded on the production of *color*, unless the arsenic is dissolved in pure water, or unless the precipitates be proved to contain arsenic, should be rejected. These remarks, however, do not apply to those cases in which clear and colorless solutions are obtained by dialysis from liquids containing arsenic in solution. The color tests may in some cases be applied to these with as much certainty as if they were applied to a solution of the poison in distilled water. The liquid tests are now generally employed rather as adjuncts to other processes, than as a direct means of detecting the poison. An exclusive reliance upon them, as *color tests*, has led to the rejection of chemical evidence on several trials, where they had been most improperly employed in the analysis of suspected liquids containing organic matter. The trial of Donnall, at Launceston, in 1817, affords a memorable lesson to the medical jurist on this subject. (Smith's *Anal. of Ev.*, p. 212.)

3. *Sulphuretted Hydrogen Test.*—The sulphide of ammonium gives no precipitate in a solution of arsenic until an acid has been added, by which property arsenic is known from most metallic poisons. On adding an acid (dilute hydrochloric acid free from arsenic), a rich golden yellow-colored precipitate is thrown down (orpiment or sulphide of arsenic.) It is better, however, to employ, in medico-legal analysis, a current of washed sulphuretted hydrogen gas, which is easily procured by adding to sulphide of iron, in a proper apparatus, a mixture of one part of strong sulphuric acid and three parts of water. The arsenical liquid should be slightly acidulated with pure diluted hydrochloric acid *before* the gas is passed into it; at least, care should be taken that it is not alkaline. The yellow compound is immediately produced if arsenic is present, and it may be collected, after boiling the liquid sufficiently to drive off any surplus gas. The precipitation is likewise facilitated by adding to the liquid a solution of hydrochlorate of ammonia. The yellow precipitate is known to be sulphide of *arsenic* by the following properties: 1. It is insoluble in water, alcohol, and ether, as well as in diluted hydrochloric acid, and vegetable acids; but it is decomposed by strong nitric and nitro-hydrochloric acids. 2. It is immediately dissolved by potash, soda, or ammonia; forming, if no organic matter is present, a colorless solution. 3. When dried and heated with two or three parts of a mixture of carbonate of soda and cyanide of potassium (see p. 307), it gives a sublimate having all the properties of metallic arsenic. Unless these properties are proved to be possessed by the yellow precipitate formed by sulphuretted hydrogen in an unknown liquid, it cannot be a compound of arsenic; and it would not be safe as a general rule to receive evidence on the point. On the other hand, when these properties are possessed by the precipitate it must be arsenic and can be no other substance.

Many objections have been taken on criminal trials to the medical evidence, founded on the application of this test; but it may be at once stated that there is no objection to the inference derivable from the sulphur-test, provided the properties of the precipitate have been determined. The objections apply only to those cases in which arsenic is said to be present, when a *yellow* precipitate is produced by sulphuretted hydrogen, and no attempt has been made to procure metallic arsenic from it. It is remarkable that cadmium should furnish, at the same time, a plausible ground of objection, both to the process by reduction from the solid state, and to the gaseous test applied to a solution of the poison. Thus the soluble salts of this metal yield, with sulphuretted hydrogen, a rich bright yellow precipitate, resembling that produced by arsenic. There are, however, these striking differences: The yellow compound of arsenic is soluble in ammonia, that of cadmium is insoluble,—the compound of arsenic is insoluble in strong hydrochloric acid, that of cadmium is perfectly soluble. With cyanide flux the sulphide of arsenic yields a volatile iron-gray sublimate of metallic arsenic, that of cadmium gives a fixed brown sublimate of cadmium oxide. An objection, on the ground of the strong similarity of cadmium to arsenic, was unsuccessfully taken to the chemical evidence given on the trial of Mrs. Burdock, at Bristol, in 1835. A persalt of tin is precipitated of a dusky yellow color by the gas; but the precipitate is destitute of all the properties of sulphide of arsenic; it is insoluble in ammonia, and yields no sublimate when heated with the cyanide of potassium flux. A salt of antimony presents no objection. A solution of this metal is precipitated of a rich orange-red (not golden yellow) color by the gas, and the precipitate yields no metallic sublimate with flux.

Marsh's Process—Hydrogen Test.—This process for the detection of arsenic was first proposed by Mr. Marsh, of Woolwich, in the year 1836. It has undergone various modifications, and has received the names of various supposed inventors or improvers, but the principle is the same, and the sole merit of the invention must be assigned to Mr. Marsh. The action of this test depends on the decomposition of arsenious acid and its soluble compounds, by hydrogen evolved from the action of diluted sulphuric or hydrochloric acid on zinc. The materials should be first proved to be free from arsenic. The apparatus is of the most simple kind, and is so well known as to need no description. The arsenic may be introduced into the short leg of the tube in the state of powder; but it is far better to dissolve it in water, by boiling it either with or without the addition of a few drops of potash or hydrochloric acid. The metallic arsenic combines with the hydrogen, forming arseniuretted hydrogen gas, which possesses the following properties: 1. Filtering-paper wetted with a solution of nitrate of silver is immediately blackened by the gas—the silver being reduced to the metallic state. Lead-paper is not changed in color unless sulphuretted hydrogen is also present. 2. It burns with a pale bluish-white flame, and thick white smoke (arsenious acid). 3. A slip of glass or of white porcelain held in the flame near the point (for not too long a time) acquires a dark stain from the deposit of metallic arsenic upon it. This deposit presents a bright metallic lustre in the centre (A, Fig. 21), a white film of arsenious acid on the outside (C), and between the two a dark ring of a pulverulent substance (B), which, when viewed by transmitted light, is hair-brown in color towards the margin, but perfectly opaque in the centre. In order to determine the arsenical nature of the deposits, the following plan may be adopted: Several of them should be received and accumulated in small porcelain capsules, held over the burning gas. To one, add a solution of chloride of lime; the deposit if arsenical is immediately dissolved. To a second, add a solution of sulphide of

FIG. 21.



Deposit obtained by Marsh's apparatus.
A. Metal.
B. Mixed deposit.
C. Arsenious acid.

ammonium: the arsenical deposit is detached, but not perfectly dissolved; yet on evaporation it yields a pale yellow film of sulphide of arsenic. To a third, add a few drops of the strongest nitric, containing some nitrous, acid. The deposit if arsenical is dissolved; evaporate the acid solution gently to dryness; carefully neutralize the residue, and add one or two drops of a strong solution of nitrate of silver. A brick-red stain or a dark-red precipitate of arseniate of silver will be produced.

This process is probably the most delicate of all those which have been devised for the detection of arsenic; but for this reason it requires the greatest caution in its application. Its delicacy has been sometimes improperly estimated by the assumed weight of the metallic deposit on glass; whereas the quantity of arsenic in one infinitesimal deposit, if transferred to the apparatus, would give no indication whatever of its presence. In operating on the poison it must be remembered that, by this process, we are dividing and subdividing the metal into a series of deposits, the weight of some of which might not be equal to the millionth part of the weight of the arsenic which is actually furnishing them. On this point a Criminal Court has been more than once misled by the medical evidence. In the *Queen* v. *Hunter* (Liverpool Spring Assizes, 1843), it was stated that the one-millionth part of a grain of arsenic might be rendered *visible* by Marsh's test; and the judge, misled by this statement, put the question to another medical witness, whether arsenic could be so removed from the stomach in three days, as that it would be impossible to discover the *one-millionth part of a grain in the body*. The deceased had died in three days after the alleged administration of a large dose of arsenic, and none was detected in the body. On this ground chiefly the prisoner was acquitted. A quantity which might be rendered visible by Marsh's process, was wrongly assumed to represent the quantity which could be detected in a dead body.

More or less arsenic is always lost during the combustion of the gas; and most of the apparatuses are so constructed, that they allow of the escape of the poisonous gas; a fact which may be demonstrated, not only by the offensive garlic odor, but by exposing a solution of nitrate of silver on filtering-paper over the open end of the tube, when a black stain will be produced. It has been objected that other substances will combine with nascent hydrogen, and when the gas is burnt, a deposit will be formed on glass, or porcelain, which may be mistaken for arsenic. A liquid containing antimony, selenium, phosphorus, sulphur, or even some kinds of organic matter, may produce a compound with hydrogen, which, when burnt, will leave a dark deposit or stain on glass. The only objection of any practical force is that founded on the presence of *antimony*, which, as a result of medicinal use, may be present in the liquids as well as in the tissues of a dead body. A current of antimonuretted hydrogen gas reduces silver from a solution of the nitrate, producing a black stain like that from arsenic, but it has not the odor of arseniuretted hydrogen. It burns with a pale lemon-yellow colored flame, and forms a white smoke (oxide of antimony). (*Guy's Hosp. Rep.*, 1860, p. 205.) The differences between the arsenical and antimonial deposits obtained by the process of Marsh are well marked. The antimonial deposit has rarely a bright metallic lustre, except when seen on the reverse side of the glass. By transmitted light, the deposit is of a smoky-black color, while that of arsenic is hair-brown. In order to distinguish these deposits we should collect a number of them from the burning gas in the interior of a small white porcelain capsule. Add a few drops of strong nitric acid. The deposit will be immediately dissolved. Evaporate gently to dryness. Moisten the dry residue with one or two drops of water, and then add a few drops of a *strong* solution of nitrate of silver. If the stain was owing to *arsenic* wholly, or in part, a brick-red colored precipitate will immediately appear. This will be more or less distinct, according to the quantity of arsenic present. Further, the red precipitate (if owing to arsenic) is entirely soluble in ammonia. A deposit of *antimony* thus treated, leaves a white residue (oxide of antimony), insoluble in water. Nitrate of silver added to it produces no colored precipitate; but if a little ammonia be brought near, either in vapor or liquid, and a solution of potash is added, a precipitate is formed, which becomes black by standing. Sulphide of ammonium dissolves the antimonial deposit immediately, and on evaporation leaves an orange-reddish colored film of sulphuret of antimony, soluble in hydrochloric acid, and insoluble in ammonia. The sulphide does not readily dissolve the arsenical deposit, but when gently evaporated, it leaves a bright yellow film (sulphide of arsenic), not soluble in hydrochloric acid, but soluble in ammonia. A solution of chloride of lime does not dissolve the antimonial deposit. Imponderable quantities of the two metals may be thus easily identified. In testing these minute films for arsenic, hydrochloric acid must not be used with the nitric, since, on evaporation, a portion, or the whole of the arsenic may be volatilized, and lost as chloride of arsenic.

A more serious practical objection to the process is that it is exceedingly difficult to procure zinc and hydrochloric acid entirely free from arsenic. The method of detecting this impurity will be explained hereafter.

Reinsch's Process.—Hugo Reinsch first published an account of this process, which originated in an accidental discovery of arsenic in hydrochloric acid in 1843. (See *Ann. d'Hyg.*, 1843, vol. 1, p. 430.) Soon after the announcement, I examined the application of it to the purposes of Medical Jurisprudence, and a full account of the results was published in the *British and Foreign Medical Review*, for July, 1843, p. 275. It has since been extensively employed in this country in medico-legal practice. It enables the analyst to trace arsenic to a minute degree in all its combinations, if we except arsenic acid and the arseniates; and in reference to these compounds, it is inferior in delicacy to the process of Marsh.

In the application of this process, the liquid suspected to contain arsenic, or the solid dissolved in distilled water, is boiled with about one-fourth to one-sixth of its volume of *pure* hydrochloric acid (proved to be free from arsenic), and a small slip of pure copper is then introduced. A slip of polished copper foil (electric copper) about a quarter of an inch square, attached to the end of a fine platinum wire, may be employed as a trial test. On boiling the metal in the diluted acid, we can at once determine whether the materials used are free from any portion of arsenic which might give rise to a fallacious result. If no free arsenic is present, the copper will acquire no tarnish or metallic deposit, but its red surface will be made much brighter. On the other hand, when arsenic is present in the liquid, even in small quantity, the polished copper acquires, either immediately or within a few minutes, an iron-gray metallic coating from a deposit of this metal. This is apt to scale off, if the arsenic is in large quantity, or if the liquid is very acid or long boiled. We remove the coated slip of copper, wash it in water, dry it, and gently heat it in a reduction tube, when arsenious acid will be sublimed in minute octahedral crystals; if these should not be apparent from one piece of copper, several may be successively introduced. When the quantity of arsenic is very small, the polished copper merely acquires a faint violet or bluish tint. Its presence may thus be indicated up to the 3000th part of a grain in a minimum of water. The deposit is in all cases materially affected by the quantity of water present, or, in other words, the degree of dilution, and sometimes it will appear only after the liquid has been much concentrated by evaporation. We are not obliged to dilute the liquid in the experiment, and there is no material loss of arsenic, as in the hydrogen process; the whole may be removed and collected by the introduction of successive portions of pure copper. We ought to be assured of the purity of the acid and copper. Arsenic is a common contamination of commercial copper in the form of foil, wire, or gauze. I have even detected it in two specimens of electrotype copper, although this is generally free from the impurity; and I have found it in what Dr. Percy describes as "best selected copper," although, according to that chemist, this should be entirely free from arsenic. (*Metallurgy*, vol. 1, p. 370.) A medical jurist must not rely upon the alleged purity of the metal, but test each sample before using it. The material best adapted for Reinsch's process is the finest copper-wire woven into a gauze, containing from ten to twelve thousand apertures to the square inch. A small piece of this, by reason of the extensive surface presented, will enable the analyst to collect a comparatively larger proportion of arsenic than would be deposited on the foil. The arsenic adheres to it with greater firmness, and the gauze will indicate by a change of color the presence of the poison, when the appearance on the foil would be indistinct.

Arsenic may be detected in copper by corroding and dissolving it in strong hydrochloric acid under free exposure to air, and distilling the dark liquid chloride of copper by a sand-bath. The distillate contains the arsenic as chloride, and this may be proved by the experiments elsewhere described. (See p. 321, *post.*)

The following process, suggested by Professor Abel, supplies a more expeditious method of detecting this impurity in copper. Boil together equal parts of a solution of perchloride of iron and strong hydrochloric acid, and while boiling introduce the slip of copper polished. If arsenic is present it is speedily indicated by a black deposit on the copper. If the metal is pure, its red color becomes more strongly marked. All the ordinary copper-foil, wire, and gauze may be thus proved to contain arsenic. This presents no obstacle to the separation of arsenic by means of it, provided the copper gauze or foil is not dissolved by the admixture of any salt such as chlorate or nitrate with the hydrochloric acid. Arsenic and copper form a solid metallic alloy. They can only be separated in the destruction of the alloy by the solution of the two metals. See "On Arsenic as an impurity in Copper." (*Guy's Hosp. Rep.*, 1860, p. 218.)

The process of Reinsch is extremely delicate; the results are speedily obtained, and are highly satisfactory. Among the cautions to be observed are these: 1, not to employ too large a surface of copper in the first instance; and, 2, not to remove the copper from the liquid too soon. When the arsenic in the liquid is in minute quantity, and this is much diluted or not sufficiently acidulated, the deposit does not take place sometimes for half an hour. If the copper is kept in for an hour or longer, it may acquire a dingy tarnish merely from the action of the acid and air. The steel-gray color of the deposit is in itself characteristic of arsenic, and may be well seen with a low power of the microscope; but there is one corroboration required. The copper with the metallic deposit upon it should be well dried, cut into small pieces if necessary, and introduced into a *dry* and perfectly clean reduction-tube. The application of a gentle heat by a spirit-lamp will cause the metallic arsenic to be volatalized as white arsenious acid, which is deposited in a cool part of the tube, in the form of octahedra or of the derivatives of the octahedron. When examined by a quarter of an inch power under the microscope these crystals may be seen and recognized by their shape up to the sixteen-thousandth of an inch in width. The smaller the crystal the more perfect in form. (See Fig. 15, p. 306.) If the copper with the deposit and the tube have not been well dried, the angularity of form may not be distinct. The crystals may be further tested by the processes already described. Such a corroboration is necessary, because the crystalline form of arsenious acid is not always distinguishable by the eye. Care must be taken not to mistake minute spherules of water, mercury, or hydrochloric acid for detached crystals of arsenious acid; and here the microscope will be found of great service. The facility of applying Reinsch's process renders it necessary that the experimentalist should be guarded in his inferences. It is not merely by the production of a deposit on copper that he judges of the presence of arsenic; but by the conversion of this deposit into arsenious acid, demonstrable by its crystalline form and its chemical properties. If a deposit take place on copper, and octahedral crystals cannot be obtained from it by heat, then the evidence of its having been caused by arsenic is insufficient. Owing to the neglect of this precaution, antimony and other substances have been occasionally mistaken for arsenic.

It is unnecessary in this place to enter into a comparison of the processes of Marsh and Reinsch, in respect to their relative powers of enabling the analyst to detect small quantities of arsenic. It may be conceded that Marsh's process will allow a chemist to detect a smaller quantity of arsenic than the process of Reinsch; but the latter, when the quantity of liquid is small, will detect the 150th or the 200th part of a grain of the poison, and this is itself a point of delicacy in analysis which, when the issues of life and death are involved, might almost suffice to justify a reasonable distrust of the resources of science. It would require considerable courage to go beyond this, and it appears to me that in a criminal case it would not be safe to depose to the presence of arsenic from Marsh's process alone, when the quantity of poison was *too small* to admit of a separation or corroboration by the process of Reinsch. Conversely the results of Reinsch's should be corroborated by those of Marsh's process. It does not at all diminish the merit of this most useful and ingenious process to say that the results which it furnishes should be corroborated by the use of some of the other tests, if it were only for the sake of preventing any plausible objections to the inference derivable from its employment. The great object of chemical evidence is not to show a court of law what may be done by the use of *one* test only, by peculiar manipulations on imponderable and invisible traces, but to render the proof of the presence of poison in the substance examined, clear and convincing. When the point of detection by Reinsch's process has been passed, then we increase the chance of fallacy to which Marsh's process is always exposed, by the fact that such very minute traces of arsenic may have existed in some of the materials used, or in the apparatus employed.

It was an over-reliance upon the extreme delicacy of Marsh's process, in researches where it admitted of no corroboration whatever, that led Orfila to assert that arsenic was a natural constituent of the human body, under the name of normal arsenic. From the results of more careful experiments he subsequently withdrew this opinion, and it is now well known that arsenic is not in any sense a constituent of the body. Under no circumstances is it found in the tissues after death, except in cases in which it has been taken by or administered to the deceased. The weakest part of the evidence in the case of Madame Laffarge (1840) was the minute quantity of arsenic detected in the body of the deceased. Orfila admitted that he had obtained, by Marsh's process, only a few deposits, so slight that they could not be weighed. He estimated the united weight at half a milligramme (.0077 gr.), or about the one hundred and thirtieth part of a grain. She was convicted of the crime of poisoning her husband with arsenic.

I am not aware that chemical evidence of the presence of arsenic in a dead body, in this country, has ever been based on a smaller quantity than in the case of Margaret Wishart. Sir R. Christison did not detect more than the *one-fortieth* part of a grain of arsenic in the coats of the stomach; but this was deemed sufficient chemical evidence, and the prisoner was condemned and executed. (*Edin. Med. and Surg. Journ.*, vol. 26, p. 23.) The smallest quantity on which I have had occasion to give evidence in criminal trials was from half a grain to a grain, estimated as the quantity actually obtained in crystals from the stomach, intestines, and tissues. (*Reg.* v. *Chesham*, Essex Lent Assizes, 1851, and *Reg.* v. *Bacon*, Lincoln Autumn Assizes, 1857.) In these two cases the accused were tried and convicted on the charge of administering poison with intent to murder. The cause of death was not here at issue.

Some experts give no assignable weight to the amount of arsenic said to have been detected, but describe it as being present in "unmistakable traces!" The danger of trusting to this loose kind of evidence in the absence of symptoms and appearances has been elsewhere pointed out. (See p. 152, *ante.*) The case of Dr. Park (*Lancet*, September 29th, 1866, p. 358, and *Law Times*, October 6th, 1866, p. 838) furnishes a strong illustration of the mischief done by the reception of such evidence.

CHAPTER XXXV.

DETECTION OF ARSENIC IN SOLIDS OR LIQUIDS CONTAINING ORGANIC MATTER—EXAMINATION OF SEDIMENT—PRECIPITATION AS SULPHIDE—DISTILLATION PROCESS—SEPARATION OF ARSENIC AS CHLORIDE FROM THE TISSUES—CONVERSION OF CHLORIDE INTO HYDRIDE—PRODUCTION OF THE METAL AND ITS TWO OXIDES—DISTINCTION OF ARSENIC FROM ANTIMONY AND OTHER METALS—REINSCH'S PROCESS FOR THE TISSUES—DISAPPEARANCE OF ARSENIC FROM THE BODY—INFERENCES FROM THE QUANTITY FOUND—ARSENIC IN THE EARTH OF GRAVEYARDS.

Arsenic in Solids or Liquids containing Organic Matter.—In testing *solids* generally for arsenic, we may employ the process of Reinsch. The solid is boiled in water acidulated with from one-fourth to one-sixth of its volume of pure hydrochloric acid, until it is either dissolved or its structure broken up. A small portion of pure copper polished is then introduced. In a few minutes, if arsenic is present—even to the extent of the thousandth part of a grain—there will be a metallic deposit on the copper, and this will yield crystals when heated in a tube. *Liquids* suspected to contain arsenic may be treated in a similar manner. In this case water is not required; the liquid is simply acidulated with one-fourth part of pure fuming hydrochloric acid. The absence of any deposit on the copper will show that arsenic is not present.

The arsenic may be mixed with the organic liquid in the form of heavy lumps or powder. The great specific gravity of this substance allows of the liquid being poured off, and the sediment collected. A large conical glass will be found most convenient for this purpose, since it admits of an examination of the nature of the sediment by a lens. In this way particles of blue coloring-matter, soot, glass, or sand, may be detected.

Facts thus observed are sometimes material as evidence in reference to proof of the possession, purchase, or administration of arsenic. Small particles of hardened fat or adipocere may be mistaken for lumps of arsenic, but they never have a crystalline appearance, while in a good light an arsenical sediment always possesses this character. By collecting the deposited substances on white blotting-paper, and passing beneath it a heated spatula, if of a fatty nature it melts, and is absorbed by the paper; if a mineral substance, it is simply dried. In the stomachs of bodies exhumed after a long interment, I have found a quantity of glittering crystals, resembling arsenic in appearance, but not in properties. They were proved to be prismatic crystals of the ammonio-phosphate of magnesia derived from putrefaction.

The sediment supposed to contain arsenic should be washed, dried, and weighed. In a strong light it always presents a well-marked crystalline appearance. In this state it may be tested by the processes elsewhere described, p. 306.

Let us assume that the poisoned organic liquid is milk, beer, or coffee, it will be necessary to determine whether any arsenic is dissolved in it. We place a portion of it in a dialyzing vessel, and immerse the bladder in distilled water (p. 154, *ante*). In a few hours the arsenic will have traversed the animal membrane, and will be found in a clear and nearly colorless solution in the water. The fluid tests may be then applied to this liquid for the detection of arsenic. They should never be applied directly to colored organic liquids. All mucous, bloody, and farinaceous liquids containing arsenic dissolved, may be thus successfully treated, and the arsenic speedily detected. It is worthy of note that, unlike some other metallic poisons, arsenic is not precipitated from its solutions by any kind of organic matter.

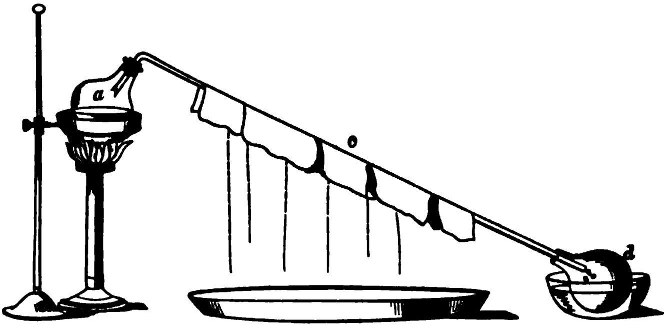
Precipitation as Sulphide.—When arsenic is contained in an organic liquid in large quantity, it may be precipitated as sulphide by a current of washed sulphuretted hydrogen. The liquid should be boiled, filtered, and acidulated with pure hydrochloric acid before passing the gas into it. When precipitation has ceased, the liquid should be again filtered, and the precipitate collected, washed, dried, and weighed. By operating on a measured portion of the solution, the amount of white arsenic may be determined by the weight of the yellow sulphide obtained —five parts by weight of sulphide being equal to four parts of white arsenic. The properties of the yellow precipitate should be verified according to the rules mentioned at p. 309, *ante*. In some cases arsenic may be present, but in a quantity too small to be precipitated as sulphide by sulphuretted hydrogen. In others the presence of certain substances may interfere with or prevent precipitation. The presence of any alkali in a liquid prevents the formation of a precipitate. For this reason the sulphide of ammonium must not be used in place of sulphuretted hydrogen. It does not precipitate a solution of arsenic until an acid is added, and acids will precipitate, from the test itself, sulphur, which has been mistaken for sulphide of arsenic. An erroneous charge of poisoning has been based on this chemical mistake.

When arsenic is found in powder, as a sediment in the fluids of the stomach, it is obvious that it must have been taken in a solid state, and although mixed with the liquid or solid, is still in an undissolved form. Arsenic, administered in a state of solution, cannot become again solid in the stomach except as a result of the perfect desiccation of the tissues. If found only dissolved, it may have been taken either in solution or in a solid form—the dissolved portion being part of the solid taken up by the fluids of the stomach, and the remainder having been expelled by vomiting and purging. This question was of importance in *Reg.* v. *Sturt*, Lewes Lent Assizes, 1863. The deceased, in this case, died from the effects of arsenic in powder, administered, it was believed, in a mince-pie. It was suggested that the poison might have been swallowed in ginger-beer, but then it could not have been in a state of solution: it must have been mechanically mixed with the liquid. The learned judge who tried this case was obviously not aware of any difference existing between the actual solution and the mechanical suspension of a solid in a liquid. The theory set up for the defence was that deceased had taken the arsenic *dissolved* in ginger-beer, and that it had reassumed the solid form in her stomach!

Distillation Process.—When the poison is in so small a quantity that it does not admit of precipitation by sulphuretted hydrogen, and no solid particles of arsenic are found in the stomach, in its contents, or in any article of food, another method must be resorted to for detecting its presence. This method equally applies to the detection of arsenic deposited as a result of absorption in the soft organs of the body, as in the liver, kidney, or heart, and to arsenic in all its forms, except the pure insoluble sulphide or orpiment. Although, after long interment, white arsenic passes more or less rapidly, into the state of yellow sulphide, as a result of chemical changes during putrefaction, the conversion is generally only partial or superficial. I have never found it to be so complete as to prevent the detection of the poison by the distillation process, in cases in which this partial conversion had taken place. The only condition for success is, that the substance, whether the contents of the stomach, food, blood, mucus, the liver, or other organ, should be first thoroughly dried by slicing it, and exposing it either to a current of warm air or to a water-bath. The dried solid should then be broken into small portions and placed in a flask or retort of sufficient capacity, with a quantity of the strongest fuming hydrochloric acid to drench it completely. The freedom of this acid from arsenic should be first carefully determined. The complete separation of arsenic from organic substances depends greatly on their perfect desiccation, and on the concentration of the acid employed. After some hours' digestion the retort or flask (*a*, Fig. 22, p. 320) containing the acid mixture—which should be of such a size that the materials should not fill it to more than one-third or one-half of its capacity—should be fitted with a long condensing tube (*c*), and then gradually heated by a sand-bath until the acid liquid begins to pass over. A metallic head, formed of a cone of tin plate or copper-foil, should be placed over the retort or flask so as to concentrate the heat and prevent condensation in the upper part of the vessel. A small flask receiver (*d*) with a loosely fitting cork may be employed to collect the product. This should contain a small quantity of distilled water, so as to fix and condense any vapors that may pass over. The receiver, as well as the condensing-tube, should be kept cool by wetting its surface with cold water diffused on a layer of bibulous paper placed over it. A perfect condensation of the distilled liquid is insured by this arrangement. The distillation may be carried to dryness, or nearly so, on a sand-bath; and it may be sometimes advisable, in order to insure the separation of the whole of the arsenic as chloride, to add to the residue in the retort another portion of pure and concentrated hydrochloric acid, and again distil to dryness. I have, however, found that portions of dried liver and stomach gave up every trace of arsenic by one distillation, when a sufficient quantity of strong hydrochloric acid had been used, and the process slowly conducted by a regulated sand-bath heat.

The liquid product may be colored, turbid, and highly offensive if distilled from decomposed animal matter. Exposure to the air for a few hours sometimes removes the offensiveness, and there is a precipitation of sulphur, or of some sulphide, without any absolute loss of arsenic. The distillate may be separated from any deposit by filtration, and, if still turbid, it may be again distilled at a lower heat to separate it from any organic matter that may have come over. The first stage of the process therefore converts the arsenic into *chloride.*

FIG. 22.



Apparatus for distilling organic and mineral substances containing arsenic.

If arsenic was present in the solid, the distillate will contain *chloride of arsenic,* which, although volatile at 270°, does not escape from a diluted solution at common temperatures. The quantity of dry organic substance used in the experiment must depend on the quantity of arsenic present, as revealed by a preliminary trial with Reinsch's process. If large, two or three drachms of the dried substance, or even less, will yield sufficient chloride of arsenic for further proceedings. For the absorbed and deposited poison, half an ounce of the dried organ, corresponding to two ounces of the soft organ, will frequently suffice; but a negative conclusion of the absence of arsenic should not be drawn from a smaller quantity than two to four ounces of the dried substance, whether liver, kidney, or heart. These tissues, it must be remembered, contain about 76 per cent. of water, so that the hydrochloric acid used will require less dilution. If oily matter should be distilled over, this may be separated by passing the distillate through a paper filter wetted with water.

I have found this process efficient for procuring a clear solution of chloride of arsenic from such different substances as starch; a cake; ordinary food; the liver and other soft organs; the scalp of the head; blood; coats and contents of the stomach and intestines; arsenical wall-papers; metallic copper; blue vitriol; and various mineral powders. I have thus discovered arsenic in two ounces of the earth of a cemetery at Boston, and in a like quantity of earth from the cemetery of Kirkby Lonsdale, as well as in the mud of the Thames and the Mersey, in spite of the presence of much oxide of iron and earthy matter. Whenever the arsenic admits of solution in hydrochloric acid, however small the quantity present, it may be readily obtained as chloride. This distillation process has the advantage of not interfering with the subsequent research in the residue for mercury, lead, copper, and other poisonous metals which do not form volatile chlorides. Arsenic may be thus separated from them; for any of the metals, if present, will be found in the residue contained in the flask or retort. Even antimony, which forms a volatile chloride, is not so readily distilled over as arsenic. (On the diffusion of arsenic and the detection of this poison in the bones, see a paper by Dr. Sonnenschein, Horn's *Vierteljahrsschrift*, 1870, p. 169.)

The distilled liquids may be preserved for future examination, or at once submitted to the second stage of the process, *i. e.*, the conversion of the chloride into *hydride* of arsenic. We should first satisfy ourselves by a few preliminary experiments that arsenic is really contained in the distilled liquid.

1. A portion may be diluted with three or four parts of water and a current of sulphuretted hydrogen passed into it. If arsenic is present in sufficient quantity a yellow sulphide will be precipitated possessing the characters elsewhere described (p. 309). If a ponderable quantity of sulphide can be obtained, the amount of arsenic present can be determined by the rule given at p. 318.

2. Another portion of the diluted distillate may be boiled with a small portion of pure copper-foil. If no metallic deposit takes place on the copper, arsenic is either not present, or in very minute quantity.

3. A few drops of the distillate undiluted may be added to a strong solution of chloride of tin, in fuming hydrochloric acid, and boiled. If arsenic is present, it is precipitated in the metallic state, of a black or brown color, according to the quantity. This reaction (Bettendorff's test) clearly distinguishes arsenic from antimony which is not reduced by the chloride of tin (p. 306).

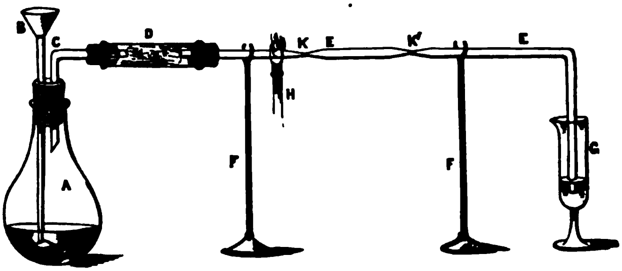
4. The distillate should be diluted with eight parts of water, and a slip of pure tin-foil introduced. If antimony is present, a black deposit of this metal takes place on the tin; if arsenic only, the tin undergoes no change. For the success of this experiment the proportion of hydrochloric acid present should not exceed one-tenth part of the liquid employed.

The first three experiments establish the presence of chloride of arsenic in the distillate; the last demonstrates the absence of antimony.

The remainder of the distilled liquid, sufficiently diluted with water to prevent too violent an action on zinc, is now introduced into a flask provided with a funnel-tube, the capacity of which must be regulated by the quantity of acid liquid to be examined. The kind of apparatus employed in this stage is represented in the engraving, Fig. 23, p. 322.

A, the flask, with funnel-tube B, and connecting-piece C; the funnel-tube should be long enough just to dip below the surface of the acid liquid. The short connecting-piece is bent at a right angle, and, like B, is carried through a closely fitting cork in the neck of the flask.

FIG. 23.



Apparatus for testing chloride of arsenic obtained by distillation.

This tube should be only long enough to go through the cork, and its open end should be bevelled off to a fine point, so that any vapor which is condensed on it may fall back as liquid into the flask. D is the drying-tube, containing fragments of chloride of calcium, secured by cotton at both ends. At the flask-end of this tube should be placed some bibulous paper, saturated with acetate of lead and dried. This has the advantage of stopping any gaseous sulphur compound, which may escape from the zinc or acid liquid. E E, a hard and not easily fusible

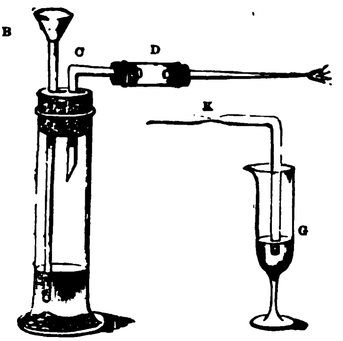
FIG. 24.

Portion of tube separated with a deposit of metallic arsenic in the contracted portion.

glass tube, free from lead, contracted in two situations K K′, to about the diameter of the tenth of an inch or less, the tube itself having a diameter of from a quarter to three-eighths of an inch. F F are supports, made of stout wire, to prevent the tube from falling when heated to redness. G is a test-glass to hold one or two drachms of a strong solution of nitrate of silver. H is a Bunsen's air-gas jet, which gives a stronger heat than a spirit-lamp, although the latter may be used.

The arrangement being thus made, the zinc and hydrochloric acid are first tested as to their freedom from arsenic. Portions of pure zinc are placed in the flask A, the parts of the apparatus are then connected, and pure hydrochloric acid, diluted with three or four parts of water, is poured into the flask by the funnel B, which operates as a safety-valve. Bubbles of air and gas speedily appear in the liquid in G, if the corks fit well, and the whole of the arrangements are air-tight. Pure zinc is sometimes but imperfectly acted on by the acid. In this case some clean platinum wire or foil may be wound round the bars of zinc, and the evolution of hydrogen will be thereby accelerated. It is, however, better that the hydrogen should come off rather slowly. If the materials are pure, the solution of nitrate of silver should undergo no change of color. The glass G should be placed on a sheet of white paper, whereby the slightest tinge of brown or black in the liquid is made perceptible. When all the air is expelled from the tube, the flame H may be applied to it at about one inch in front of a contraction of the glass, as indicated in the engraving, and the glass heated to redness. No metallic deposit should take place at K. If the materials are quite pure, there will be no deposit, and the transparency of the glass tube at K will be unchanged. From a quarter to half an hour will be sufficient for this experiment. This is the best and surest method of determining whether the zinc and hydrochloric acid are entirely free from arsenic. The purity of the hydrochloric acid may be also tested by boiling a portion of it with a concentrated solution of chloride of tin.

FIG. 25.



Tube apparatus for testing a small quantity of liquid containing chloride of arsenic.

If it gives a brownish-black precipitate, or acquires a brown color, this is a proof that it contains arsenic. Zinc converted into a salt by any pure acid may be tested in a similar manner. On the same principle, hydrochloric acid may be obtained free from arsenic by distilling it over fragments of pure tin.

A portion or the whole of the distillate is now gradually added to the acid liquid in the flask by means of the funnel-tube, taking care that it is never more than one-third full. The first indication of the presence of arsenic will be manifested by the silver solution becoming brown, and finally black, a dense precipitate of metallic silver resulting from the chemical changes. If it should become very suddenly black and flaky, the presence of sulphur may be suspected. This will be further indicated by a change of color in the lead paper in D. Pure arseniuretted hydrogen does not alter the color of this paper. When the silver-solution is nearly blackened, the flame may be applied as indicated in the engraving, and kept steadily at this point. At a red heat, visible in daylight (1200°), arseniuretted hydrogen is decomposed, and metallic arsenic is deposited; but being a volatile metal, it is carried onward by the hot current of gas, and forms at first a brown and then a black metallic mirror at K, *i. e.*, in the contracted part of the tube, which is cool, a little in front of the part heated. When a sufficiently thick deposit is obtained, the flame may be applied to the tube about an inch in front of K. Thus as many deposits of metallic arsenic may be procured as there are contractions in the glass tube.

The silver solution is allowed to become saturated with the gas. Any escape of the gas from the glass, or by leakage from any of the junctions of the apparatus, is at once indicated by holding near to the spot filtering-paper wetted with a solution of nitrate of silver. This is instantly blackened by the escaping gas. The glass with the silver solution is then removed, the end of the tube well washed, or another tube substituted for E E, and this is allowed to dip into about one drachm of the strongest fuming nitric acid, containing much nitrous acid in a test-glass similar to G, or into a small porcelain capsule. After a time, the acid loses its color, and the metallic arsenic of the gas is converted into arsenic acid, which may be obtained for the purpose of testing by evaporation.

The further examination of the products is a very simple process. 1. The silver solution contains arsenic in the state of arsenious acid dissolved, with some nitric acid and the excess of nitrate of silver. By one or two filtrations it is obtained colorless and clear. A weak solution of ammonia is then added to it, and yellow arsenite of silver is at once precipitated (see p. 309). This result shows the presence of arsenious acid, the first or lower oxide of arsenic. 2. The nitric acid, into which the hydride has been passed, is evaporated to dryness in a small porcelain capsule. One or two drops of water are added to the residue, with a drop of weak ammonia if it should be very acid. A strong solution of nitrate of silver is then added to it; arseniate of silver, of its well-known brick-red color, is immediately produced. This demonstrates the presence of the second or higher oxide of arsenic; and the proof of the presence of arsenic in the distillate, and, therefore, in the substance from which it was obtained, may be considered as absolutely complete.

The solution of nitrate of silver and fuming nitric acid to receive the gas may be dispensed with, since two of the metallic deposits obtained by heating the hydride may be respectively converted into arsenious and arsenic acids by the processes elsewhere described, and thus tested; but the use of the nitrate of silver solution prevents the escape of any of the hydride, and does not interfere with the reduction of the hydride by heat. One of the deposits may be hermetically sealed for production in evidence.

With these results the evidence of the presence of arsenic may be considered as conclusive. The poison is obtained by this process, not only in its pure metallic state, but in the distinct forms of its two well-known oxides,—arsenious and arsenic acids. Any demonstration beyond this is superfluous. It will be observed that Reinsch's process is here employed merely as an adjunct to Marsh's process in an improved form, in which the burning of the gas is unnecessary. The arsenic by distillation is first converted into chloride, the chloride into hydride, and the hydride into the metal and the respective oxides. In the different stages of this operation, all other metals, excepting antimony, are entirely excluded, and this, under the circumstances, may be easily distinguished from arsenic, not only by Bettendorff's test, but by the action of a solution of nitrate of silver. A black deposit takes place, but no arsenious acid is formed.

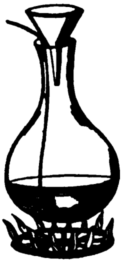
The zinc which has been used for one experiment is not fitted for use in a second. M. Blondlot has discovered that in the usual method of operating on distilled zinc with hydrochloric or sulphuric acid, only moderately diluted, a blackish-brown flaky compound is formed, which detaches itself from the zinc and floats in the acid liquid. He finds this to be arsenic in the form of a solid and insoluble hydride. Concentrated or diluted acids do not act upon it in the cold, but at a boiling temperature these acids decompose it. Nitric acid and chlorine rapidly dissolve it in the cold. Heated in a dry tube it is resolved into arsenious acid and water. Nascent hydrogen has no action upon it unless some organic matter is present, when it is instantly converted into arseniuretted hydrogen. It commonly attaches itself to the zinc employed in the experiment; by exposure to air it is gradually transformed into arsenious acid; and thus a bar of zinc which does not show the presence of arsenic on its withdrawal from the acid, may become coated with oxidized arsenic, as a result of exposure, so as to lead to a serious fallacy if used in another experiment.

Dr. Schneider, of Vienna, was the first to suggest, in 1851–2, a method of extracting arsenic from organic matter as a volatile chloride. For this purpose he employed strong sulphuric acid and dry common salt. His process is well known under the name of Schneider's process. It was described in the 5th edition of my *Manual of Medical Jurisprudence*, 1854, p. 80 (see also *Pharmaceutical Journal*, July, 1853, p. 38, and *Guy's Hospital Reports*, 1870, p. 240). It has the disadvantage of introducing much mineral matter into the substance distilled, as well as of producing a large amount of froth, and thus embarrassing the operation. The modification of it, above described, in which pure hydrochloric acid alone is required, will be found more convenient in practice.

The late Dr. Penny, of Glasgow, first employed distillation with hydrochloric acid for the detection of absorbed arsenic in the tissues in 1852; but as he mixed water with the acid, no chloride of arsenic was obtained in the first portions distilled. Dr. Odling subsequently applied it to the stomach of a dog; but he found the residue of the distillation to contain arsenic, and the process in his opinion did not present any advantages to compensate for the increased trouble attending it. (*Guy's Hospital Reports*, 1855, p. 294.) The process was subsequently successfully employed for medico-legal purposes by Dr. Penny and Sir R. Christison, in the case of L'Angelier, in 1857. (*Report of the Trial of Madeline Smith*, Edinburgh, 1857, p. 61.)

Reinsch's process alone may be employed for detecting arsenic deposited as a result of absorption, in the liver, kidneys, or other organs. About four ounces of the recent organ, or more, if necessary, cut into very thin slices, should be boiled in a flask in a mixture of one part of pure hydrochloric acid and two of water, until the structure of the organ is broken up. The flask may be of the shape represented in the annexed engraving (Fig. 26), and heated either by a sand-bath or through a layer of iron gauze. A small glass funnel should be placed in the neck of the flask. This receives and condenses the vapor, which falls back into the flask. By this arrangement the boiling may be continued for a long time, without material loss by evaporation. The flask should not be more than half full, and heated gently until all froth is ex-

FIG. 26.



Flask employed in the analysis of substances by Reinsch's process.

pelled. A fine platinum-wire, having a small piece of pure copper-foil or gauze attached to it, should be immersed in the liquid when boiling. This enables the operator to remove the copper and examine it at intervals, after immersing it in distilled water. If it is much coated with a metallic deposit, larger portions of copper-foil may be successively introduced until the liquid is exhausted. The copper should be boiled in alcohol or ether, to remove any adhering organic matter, and the deposit on the metal may then be tested by the methods described at page 313. It is remarkable for what a length of time the copper retains the arsenic deposited upon it. Some copper-gauze on which arsenic had been deposited was examined after twenty-five years, and, although much changed in appearance by exposure, it yielded a perfect sublimate of octahedral crystals (*Reg.* v. *Jennings*, Reading Lent Assizes, 1845). This process is well adapted for the detection of arsenic in the urine and saliva eliminated from the living body, and in all liquid articles of food.

It might be supposed that arsenic would escape as chloride in this method of operating, but when hydrochloric acid is diluted with the proportion of water recommended, little or no volatile chloride is distilled over. In reference to the recent organs, a larger proportion of acid is used, because three-fourths of the weight of the animal substance really consist of water.

The delicacy of this process may be estimated from the following experiment: In January, 1852, six ounces of a thick turbid fluid were taken from the stomach of a dog supposed to have been poisoned. As there was no poison present, the copper-gauze coming out of the liquid clean and without any deposit upon it, five drops of a solution of arsenic, equivalent to six-hundredths (.06) of a grain of the poison, were put into the mixture, and the whole was well stirred. In ten minutes the copper was coated of a steel-blue color, and crystals were obtained from it by heat. The 16th part of a grain of arsenic was here detected, in about forty thousand times its weight of a complex organic liquid.

For the detection of arsenic in the tissues, various processes have been suggested. Fresenius and Babo destroy the organic matter by hydrochloric acid and chlorate of potash, and advise a series of proceedings of a most minute and elaborate kind. In fact, this mode of detecting arsenic may be designated an exhaustive process. It provides for the exclusion of lead, bismuth, mercury, copper, tin, antimony, and other metals; but in thus excluding many bodies which are never likely to be found, it incumbers the investigation with the employment of so many chemicals, that a question might fairly arise whether arsenic had not been actually introduced into the organic matter during the operation. I have known only one case in which it has been medico-legally employed in this country, that of *Reg.* v. *Wooler* (Durham Winter Assizes, 1855), and there fortunately the proof of death from arsenic was so clearly made out from other facts, that it was unnecessary to make this elaborate mode of testing a subject of cross-examination. The reader who is curious about this process, the complication of which, according to Orfila, surpasses all credibility, will find the details in Orfila (*Toxicologie*, vol. 1, p. 515, Otto, *Ausmittelung der Gifte*, p. 34, 1856, and Böcker, *Die Vergiftungen*, 1847, p. 88.)

It is unnecessary to notice the ancient processes of carbonization of the organs with sulphuric acid, or deflagration with nitre. If the observations of M. Blondlot are correct, this and the chloride process have been the source of great and unsuspected errors in medico-legal analysis. (*Ann. d'Hygiène*, 1864, vol. 1, p. 152.)

It is important, in reference to the presence of absorbed arsenic in the *tissues*, to observe that it may be found at an early period, when it is either absent or only doubtfully present in other parts. In a case referred to me in May, 1854, the deceased, Burton, died within *four hours* after he had been attacked with symptoms of poisoning with arsenic. Arsenic was found in small quantity in the stomach, duodenum, and rectum. It was also detected in the liver and spleen; and the proportion found was greater in the latter than in the former organ. In November, 1861, a man died from the effects of arsenic in the most acute form, soon after his admission into Guy's Hospital. He had swallowed unknowingly a large dose of the poison in water. His wife left him at 1.30 P.M. quite well; during her absence he swallowed the poison, and on her return at 4, she found him very ill and suffering from vomiting and purging. He was brought to the hospital, and died soon afterwards. Barely *three hours* could have elapsed from the time at which the poison was taken until his death. There were the usual appearances in the stomach, intense inflammation, especially at the pyloric end; and gritty portions of arsenic, mixed with masses of coagulated mucus and false membrane, were found in the contents. The intestines were inflamed, and portions of arsenic were discovered as low as the cæcum. Arsenic was found abundantly in the stomach, and a comparatively large quantity of the poison was detected in half an ounce of the dried liver, as well as in the spleen and kidney. Hence it is obvious that the poison may be rapidly absorbed and copiously deposited in these organs within the short period of *three* hours. In the cases of the Atlee family, referred to me by Mr. Carter, coroner for Surrey, in January, 1854, the body of the mother was exhumed after a month. Arsenic was *not* found in the *stomach* or *bowels*, but it was readily detected in a small portion of the *liver*. The poison had probably been taken several days before death. The fact is of considerable importance in relation to a medical opinion of the presence or absence of poison in a dead body. It is commonly the practice to confine an analysis to the stomach and bowels only; and when no poison is found therein, to report that no poison exists in the body, and to refer death to natural causes. It is clear, however, from the above case, that such an opinion might be erroneous unless the liver or spleen had also undergone a chemical examination. In preserving viscera for analysis, a portion of the liver should therefore always be set apart for examination. If the person has lived fifteen or sixteen days after having taken the poison, no trace may be found in the tissues or in any part of the body. Orfila long since expressed this opinion from his experiments on animals; its correctness has been strikingly confirmed by the case of Dr. Alexander, who died in *sixteen days* from a large dose of arsenic taken by mistake in arrowroot. The late Dr. Geoghegan, who was deputed to make an analysis of the stomach and other viscera in this case, found no trace of the poison, either absorbed or unabsorbed, in any part of the body, which he examined. (See *Med. Times and Gazette*, April 18, 1857, p. 389.) It is the more necessary that the fact of entire elimination should be remembered, because it has been most erroneously impressed on the public mind, that no person can die from poison, except the poison be found by chemical analysis in the body after death. On some trials which have excited much public notice, men of repute as toxicologists have greatly added to the difficulties of medical evidence, and have embarrassed medical witnesses, by affirming that if death had really taken place from poison, it should always be found in the body. If this is untrue with respect to arsenic, it is necessarily untrue with regard to poisons less easy of detection in minute quantities.

In the case of the *Queen* v. *Williams* (South Wales Circuit, July, 1863), the late Mr. Herapath, of Bristol, confirmed by his evidence the observations of Dr. Geoghegan, and proved that there is a limit to the period for the detection of arsenic in the body of a person who has died from the effects of this poison. A woman was charged with the murder of her husband by administering to him arsenic. The evidence left no doubt that deceased had suffered from the usual symptoms of poisoning, namely, inflammation of the stomach and bowels, numbness of the limbs, and other symptoms. For some days before the deceased's death, owing to his food having been prepared for him by his daughter, the symptoms abated; but he ultimately died from exhaustion on the fifteenth day. Mr. Herapath examined the viscera, but found no arsenic. Neither in his reading nor in his experience had he known arsenic to have been detected so long as fifteen days after its administration. As no poison was discovered in the body, the prisoner was acquitted on the charge of murder, but found guilty of the intent. (*Lancet*, July 11, 1863, p. 47.) In the case of Soufflard, a large dose of arsenic had been taken; but according to M. Legroux, no trace of the poison existed in the stomach or in the ulcerated portions of the bowels. (*Union Médicale*, June 30, 1850.) Dr. Otto met with a case of death from arsenic within twenty-four hours, with the usual symptoms and appearances, but no arsenic could be found in the contents of the stomach. In this case the liver and other organs were not examined. (Horn's *Vierteljahrsschrift*, 1865, vol. 1, p. 175.) This observation therefore refers to free and not to absorbed arsenic. Arsenic has been sought for, and not detected, at shorter periods after death when there was a very strong suspicion that the poison had been taken; but it is highly probable that in these cases there was little or no arsenic remaining in the bodies at the time of death.

The longer a person has survived after taking this poison the less probable is it, *cæteris paribus*, that arsenic will be found in the remains. With respect to its detection in the stomach and bowels, if the vomiting and purging have been violent and the person has survived some days none may be found. It is singular, however, to notice with what tenacity the mineral sometimes adheres to the mucous membrane in spite of vomiting and purging. In the case of the Duc de Praslin, who died in six days from a large dose of arsenic, some portion was still found in the intestines (*Ann. d'Hygiène,* 1847, p. 402); and in a case which was the subject of a criminal trial at the Leicester Autumn Assizes in 1860 (*Regina* v. *Holmes*), Mr. Lankester informed me that arsenic was detected in the intestines, although the deceased had survived the effects of a large dose for seven days and had suffered from the usual symptoms.

The preservative effect of arsenic on the solid organs of the body has been already noticed (p. 127). Dr. L. Penard has directed attention to a fact connected with the presence of arsenic in a dead body, not hitherto noticed by medical jurists. Under a suspicion of poisoning with arsenic, ten bodies were exhumed in the district of St. Colens, in 1869. Dr. Charbonnier, who was charged with this duty, found that arsenic was present in quantity in two of the bodies which had been well preserved. There was no offensive smell of putrefaction about them, but a remarkable alliaceous odor like that of phosphorus. (*Ann. d'Hyg.*; 1873, Juillet, p. 186.) This was attributed to the probable escape of arseniuretted hydrogen as the result of decomposition, and thereby a loss of arsenic. On this point I may observe that in several cases of exhumation in which arsenic was discovered in the bodies, no odor of the kind was perceptible. Further, it may be stated that phosphuretted hydrogen has a similar odor, and I have observed this in remains in which no arsenic was detected.

When arsenic is discovered in the stomach mixed with food, it does not necessarily follow that it has been administered in that particular article of food. Should the person have partaken of liquid food, such as milk or gruel, subsequently to the swallowing of arsenic, these fluids will necessarily acquire an arsenical impregnation from the poison already contained in the stomach. The patients may have taken the arsenic in one kind of food, when another and an innocent description of food given subsequently, might thus be inadvertently pronounced to have been the vehicle. (See on this point the case of Ann Merritt, *London Med. Gaz.*, Aug. 16, 1850, p. 291.) Among the chemical facts deserving the notice of the analyst is this: arsenious acid reacts upon Trommer's test like grape-sugar, and this substance might thus be wrongly pronounced to be present in place of arsenic.

It need hardly be observed that the *quantity of arsenic found in the stomach* or other organs can convey no accurate idea of the quantity actually taken by the deceased, since more or less of the poison may have been removed by violent vomiting and purging as well as by absorption and elimination. A large quantity found in the stomach or bowels indicates a large dose, but the finding of a small quantity does not prove that the dose actually taken was small. Notwithstanding these very obvious causes for the removal of a poison from the body, there is a strong prejudice among lawyers that the chemical evidence is defective unless the quantity found is sufficient to cause the death of another person. It would be just as reasonable, in a case in which a man has been killed by a discharge of small shot, to insist upon a failure of proof of the cause of death because only a single pellet has been found in the body. The value of chemical evidence does not depend on the discovery of any particular *quantity* of poison in the stomach—it is merely necessary that the evidence of its *presence* should be clear, distinct, conclusive, and satisfactory. At the same time a reasonable objection may be taken to a dogmatic reliance upon the alleged discovery in a dead body of minute fractional portions of a grain, or "traces;" and, considering the great liability to fallacy from the accidental presence of arsenic in the articles used for analysis, the chemical evidence in the French case of Madame Laffarge (1840), was of a most unsatisfactory kind, and should have been rejected by the court. (See p. 316.)

When the poison is really present in the stomach at the time of death, it does not easily disappear, and it may, therefore, be discovered for a long period after interment. White arsenic becomes slowly changed into yellow sulphide by the evolution of sulphuretted hydrogen in the putrefaction of the stomach or its contents. It frequently forms a deep yellow stain through the coats, and appears on the external surface. I have thus found it converted into sulphide twenty-eight days after interment (*Reg.* v. *Jennings*, Berks Lent Assizes, 1845); but the change may take place in a much shorter period. In recent cases the conversion is, however, in general only partial, as white grains may be often seen in the yellow mass. In the cases of the Cheshams (Essex Lent Assizes, 1847), which I was required to examine, the coats of the stomachs were in both instances deeply dyed with large patches of yellow sulphide nineteen months after interment. This change of color from white to yellow is not always met with, even in bodies which have been buried for a year, or longer. (*Guy's Hosp. Reports*, Oct. 1850, p. 206.) Care must be taken not to confound stains produced by bile on the stomach or intestines with those caused by sulphide of arsenic.

Arsenic has been detected in a body at the end of three years (Galtier, *Toxicologie*, i, 370), and even after seven years. (*Devergie*, i, 313.) Wöhler is stated to have detected it in the bodies of two men seven years and six months after burial; this, however, was absorbed arsenic, and the process pursued was incineration of the soft organs by nitre. (*Ann. der Chim. und Pharm.*, liii, 141; *Chemical Gaz.*, 1845, 192; *Med. Gaz.*, vol. 35, p. 655.) In another case it is reported to have been detected after ten years. The body had become reduced to a skeleton. A confession was made by some of the parties concerned in the murder: corroborative evidence was sought for, and as it was asserted that a large dose of arsenic had been given, and the person had died in twenty-four hours, the remains of the skeleton, identified as that of the deceased, were submitted to chemical examination, when arsenic was readily detected. The examination of another skeleton found near that of the deceased led to a negative result; no arsenic was found. The jury were satisfied with this evidence, and returned a verdict of guilty. (*Journal de Chimie Méd.*, Fevrier, 1847, 82.) The particulars of the analysis are not given, nor is it stated whether the earth around the skeleton did or did not contain arsenic. Dr. Glover discovered the poison in the body after twelve years (*Lancet*, July 9, 1853, p. 41); and Dr. Webster, U. S., in the remains of a body after fourteen years' burial in a tomb (*Med. Gaz.*, 1849, vol. 43, p. 894).

When the contents of the stomach are not allowed to drain away, the poison may be detected after a very considerable period. A person died from the effects of arsenic on the 21st of February, 1824,—the poison was at the time easily found in the contents of the stomach: these were kept for upwards of thirteen years loosely covered, and arsenic was then as readily detected in them as in the first instance,—whether Marsh's or Reinsch's process, or sulphuretted hydrogen, was employed. Except by the entire destruction of the body in a case of arsenical poisoning, a criminal cannot now defeat the objects of a chemical investigation. Dr. Schäffer met with a case in which an attempt was made by the accused to destroy the dead body by fire. A woman had suffered from symptoms of poisoning by arsenic, and died in about eight hours. Notice was given to the husband that there would be an inspection of the body, and on the next night his house was found in flames. The dead body of the wife was so burnt that it could scarcely be recognized. The stomach, however, had not been entirely destroyed, and in the shrivelled remains of this, some particles of solid arsenic were found. The facts were clearly proved against the husband. (Casper, *Vierteljahrsschrift*, B. 14, H. i, Juli, 1858, p. 88.)

In examining the decomposed remains of a body when mixed with the earth of the grave, care must be taken that the traces of arsenic occasionally found in ochreous soils, are not mistaken for arsenic criminally administered to the deceased. In those soils in which iron pyrites are common, arsenic may be present as a native constituent; but it has hitherto been found only in small proportions and in a form entirely insoluble in water, so that water could not dissolve it and transport it into a body lying in a grave. Iron pyrites contain arsenic, and by exposure to air they become disintegrated, and the arsenic, in insoluble combination with iron, is diffused through the soil. Arsenic has thus been found as a constituent of the earth of certain cemeteries in Norfolk, Lincolnshire, and other parts of England. In 1862, I had occasion to examine the remains of a man named Peter Mawer, whose body had been buried for a period of eight years in the cemetery of Boston, Lincolnshire. A quantity of the earth from the grave was examined at the same time. No arsenic was found in the remains, which were sodden and adipocerous owing to the coffin having given way, so that water and earth had penetrated into it in all directions. A few ounces of the earth of the grave, separately examined by distillation with hydrochloric acid, yielded metallic arsenic; but the earth, boiled in water, yielded no arsenic. If in any case a dead human body would have been likely to acquire an arsenical impregnation after burial in an arsenical soil, it would have been on this occasion. The negative result is simply confirmatory of facts observed by others, that soluble arsenic is not found in the earth of cemeteries, and therefore any objection to the chemical detection of arsenic in an exhumed body, has no real foundation. Orfila's experiments had already shown that the remains of bodies buried in arsenical earth for a period of three months, did not acquire thereby any arsenical impregnation. (*Acad. of Med.*, June 29, 1847; *Gaz. Méd.*, July, 1867, p. 535; also *Ann. d'Hyg.*, 1847, vol. 2, p. 194.)

If a medical expert, owing to neglect or inattention, mixes the earth and human remains together and acts upon them with powerful acids, arsenic may be found; and unless the soil of the grave has been separately examined and found free from arsenic, a mistake may be made.

Flandin found arsenic in the earth of certain graveyards in France, and in one instance he estimated that the proportion of arsenic did not exceed the twentieth part of a grain in a pound of earth, or one grain in twenty pounds! The arsenic was in all instances insoluble in water. (*Traité des Poisons*, vol. 1, pp. 429, 442; Galtier, *Toxicologie*, vol. 1, p. 368; and *Lancet*, August 17, 1844, p. 638.)

The reader will find cases in which doubts based upon the origin of the arsenic detected in a decomposed dead body have led to the rejection of chemical evidence. (Flandin, *Traité des Poisons*, vol. 1, pp. 674, 683.) When ordinary precautions are taken, a difficulty of this kind cannot, however, present itself in practice. A body buried in a coffin in this country is rarely so far decomposed as to become covered by the soil from the destruction of the wood in a period shorter than from seven to ten years; and until such a complete destruction has taken place, it is not easy to perceive how the presence of an *insoluble* arsenical compound, as a natural constituent of the soil, can present any objection to the results of a careful chemical analysis.

This question at one time acquired some importance in this country from the result of the trial of Elizabeth Johnson for poisoning her husband with arsenic (Liverpool Lent Assizes 1847). The accused, concerning whose guilt, morally speaking, there could be but little doubt, appears to have owed her acquittal entirely to the assumption that arsenic in a soluble form might have found its way into a dead body through a crack in a coffin, although it had not been shown that the soil of the churchyard where the body was buried contained any trace of arsenic, either soluble or insoluble! Still, so far was this scientific question carried, that the assumption of arsenic being present in the soil was allowed in favor of the prisoner; the onus of disproof was thrown on the prosecution.

The deceased, it appears, died on Dec. 3, 1846. The body was exhumed, after three months' interment, on March 9, 1847; and the late Mr. Watson, of Bolton, examined the viscera. This gentleman clearly detected arsenic in the liver, intestines, heart and blood, kidneys, gullet, and tongue, and in the muscular substance of the right thigh. The largest proportion of poison was derived from the liver, kidney, and intestines; and the smallest quantity, amounting to only an exceedingly slight trace, was detected in the heart and blood. Mr. Leigh, a medical witness who was present at the exhumation, stated that there was a large quantity of fluid blood in the body. When asked by the learned judge (now deceased) who tried the case, to account for this, he said he thought it might have arisen from the penetration of water, as the grave was wet, and the coffin was split from one end to the other. The wood of the coffin was thin, and it had apparently cracked from the superincumbent weight of earth. The judge then asked, "Supposing there was arsenic in the soil of the churchyard, was it not possible for some of that arsenic to be washed into or introduced into the body along with the water?"—to which Mr. Leigh replied, he thought it was possible. This statement, so materially affecting the chemical evidence, was allowed to pass to the jury as a probable mode of accounting for the presence of arsenic, not in the fluid blood only, but in the liver, kidneys, intestines, and even the tongue, œsophagus, and muscles of the thigh! The first intimation which Mr. Watson received of the mode in which his chemical evidence would be applied, was in the charge of the learned judge to the jury; and there can be no doubt that owing to there being no evidence of the presence of arsenic in the body before interment, the jury referred its discovery three months afterwards to the series of assumptions above detailed.

No questions on the subject were put to Mr. Watson at the trial, or he would have fully explained the matter. He subsequently procured a quantity of earth from the churchyard in which this body was buried, and, on a careful examination, he did not detect in it a trace of arsenic, either in a soluble or insoluble form.

The arsenic found under these circumstances has been improperly called *cemetery arsenic;* but it need hardly be observed that the poison may be equally found in any soil containing iron pyrites.

When food or vomited matters containing arsenic have been thrown on the earth, this question may incidentally present itself as an objection to chemical evidence. In a case which was the subject of a trial in France (*Affaire Malaret*, May, 1846), it appeared that the wife, who was charged with the murder of her husband, had thrown some of the vomited matters out of a window. The ground beneath was examined, and arsenic was found in the earth immediately under the window, and in a smaller quantity for a circuit of more than two yards. Beyond this space the earth did *not* contain arsenic. The poison was detected in the tissues of the body, which had been for some time buried, and exhumed for the analysis. The earth of the graveyard was examined, and contained no arsenic. (*Gaz. Méd.*, 20 Juin, 1846, p. 498; and *Ann. d'Hyg.*, 1847, vol. 1, p. 400.) A similar question arose in the case of *Reg.* v. *Lucas and Reeder* (Cambridge Lent Assizes, 1850). The deceased had died from the effects of arsenic. One of the sources of proof was derived from the analysis of some earth taken from a garden-path, on a spot where the deceased had been seen to vomit in the early part of her illness. I found arsenic in a few ounces of this earth, in a soluble form, and in rather large proportion. In earth taken from the path at a distance from this spot, no arsenic was found, either in a soluble or insoluble form.

CHAPTER XXXVI.

COMPOUNDS OF ARSENIC—ALKALINE ARSENITES—FOWLER'S SOLUTION—ARSENICAL SHEEP-WASHES—ARSENITE OF SODA—ARSENITE OF COPPER—ACCIDENTS CAUSED BY SCHEELE'S GREEN—USE IN CONFECTIONERY—ARSENICAL WALL-PAPERS—THEIR EFFECTS ON HEALTH.

ARSENITE OF POTASH—FOWLER'S SOLUTION.

ALL the compounds formed by arsenious acid with the alkalies are poisonous. Those of potash, soda, and ammonia are soluble in water, and, therefore, act with more energy. The **ARSENITE OF POTASH** is used in medicine, and is well known under the name of **FOWLER'S MINERAL SOLUTION**, or Tasteless Ague Drop. It is made by boiling arsenious acid with carbonate of potash, the latter being in slight excess, and it is colored with compound tincture of lavender. In the preparation adopted in the British Pharmacopœia there are four grains of arsenious acid in a fluid-ounce (or eight fluid drachms) of the solution ($\frac{1}{120}$th gr. in one minim); but its real strength may be affected by impurities in the arsenious acid employed. The action of this liquid as a poison, in large doses, is in all respects analogous to that of arsenious acid. It acts more energetically by reason of the poison being in a perfect state of solution.

Symptoms and Appearances.—There is, so far as I know, only one instance recorded in which this solution has destroyed life. (*Provincial Journal*, June 28, 1848, p. 347.) A woman took half an ounce of the solution (= two grains of arsenic) in divided doses, during a period of five days. There was no vomiting or purging, but after death the stomach and intestines were found inflamed. The solution is said to be much used by the poor in the Fen districts of Cambridgeshire, as a preventive of ague. It has occasioned symptoms of poisoning when given in an overdose, but I have not heard of any case proving fatal. This domestic use of arsenic in these districts may, however, account for the occasional detection of "traces" of arsenic in a dead body, irrespective of criminal poisoning.

That Fowler's Solution is a powerful agent, and that the boundary between its medicinal and poisonous action is very slight, will be sufficiently proved by cases elsewhere reported.

There is one form of poisoning by an alkaline arsenite which it is desirable to point out. A mixture of arsenic and carbonate of potash with soft soap and tar-water is largely used in agricultural districts for killing the fly in sheep. There is no doubt that a mixture of this kind is injurious to sheep, unless carefully made and properly used. Mr. Tuson gives the following proportions for a sheep-dip: White arsenic, in powder, and carbonate of potash, of each eight to ten ounces;

water, twenty gallons; to be boiled for half an hour. This quantity is sufficient for twenty sheep. Soft soap and flowers of sulphur are sometimes added. (*Veterinary Pharmacopœia*, 2d edit., 1874, p. 150.) There is reason to believe that this proportion of arsenic has been often greatly exceeded, and the dippers and the sheep have suffered in consequence. In *Black* v. *Elliott* (Newcastle Lent Assizes, February, 1859), damages were claimed for the loss of 850 sheep said to have been poisoned by dipping them into a strong arsenical solution. The jury were satisfied with the evidence proving carelessness and neglect in the employment of the wash, and found a verdict for the plaintiff, with £1400 damages. In *Smith* v. *Barker* (Bury Summer Assizes, 1870), it was proved that eighteen sheep had been killed by the use of an arsenical wash; but as there was contributory negligence on the part of the plaintiff's workmen, in making the solution too strong, a verdict was returned for the defendants, the manufacturers of the arsenical compound. In June, 1853, a woman swallowed a quantity of sheep-dipping liquid. She soon afterwards experienced severe pain in the stomach, nausea, and violent vomiting, followed by purging, great heat in the throat and abdomen, with intense thirst. She died in twenty-four hours after taking the poison. The mucous membrane of the stomach presented a few patches of inflammation, but was of a pale bluish color in other parts. The lining membrane of the gullet, at the part where it entered the stomach, was of a bright pink color. The duodenum and upper part of the small intestines were highly inflamed. A portion of the liquid which deceased had taken was found to be highly alkaline, and smelt strongly of tar. It was a saturated solution of arsenite of potash, with a large proportion of carbonate. When paper impregnated with the liquid was burnt a white smoke was evolved, which, when received on a cool surface of glass, gave the usual indication of arsenious acid with ammonio-nitrate of silver. The symptoms and appearances were similar to those observed in poisoning by arsenious acid.

Dr. Mitchell met with a case in which a mixture of arsenic and soft soap, used locally, produced all the well-marked effects of poisoning with arsenic, as well as an intense local action. A man applied this mixture to his scrotum and armpits for the purpose of killing pediculi. In twelve hours he began to feel a stiffness in the neck, and a slight difficulty in swallowing. The cuticle of the scrotum peeled off, leaving the cutis inflamed and bleeding. There was great thirst, with headache, and a sensation as if the hair was being pulled up by the roots. There was irritability of the stomach, with vomiting, purging, and pain on pressure. He said that he felt as if his bowels were on fire. Under treatment this man recovered. (*Med. Times and Gaz.*, Dec. 10, 1853.) Shepherds who have used this solution for dipping sheep, have occasionally suffered severely from symptoms of irritant poisoning as well as from eruptions on the skin.

Orfila refers to a singular case of poisoning by a compound arsenite of potash and lime in a solid form (*i. e.*, as a soap), in which the most marked nervous symptoms (trismus) appeared in three-quarters of an hour; the individual recovered. (*Toxicologie*, vol. 1, p. 449.)

Fatal Dose.—The medicinal dose of Fowler's Solution is from four to ten minims twice a day. It is common to commence with four to five minins, and gradually increase the dose. The late Dr. Pereira states that fifteen minims had been taken three times a day for a week without ill effects; and Dr. Mitchell, of Ohio, has given from fifteen to twenty drops three times a day in intermittents. (*Materia Medica*, vol. 1, p. 718.) In some persons there is a strong idiosyncrasy with respect to arsenic; and even smaller doses than those commonly prescribed can hardly be borne without causing alarming symptoms. A case was reported in the *Pharmaceutical Journal* for 1845, in which one drachm of the solution (equal to half a grain of arsenic) was taken with comparative impunity.

According to Mr. Bullock, the pharmacopœial preparation is not a true arsenite of potash, but a solution of arsenious acid in carbonate of potash, with a minute trace of the arsenite. (*Lancet*, Dec. 21, p. 674.) The uncertainty of its composition may possibly account for the variable effects produced by this liquid. Mr. Hunt, who has largely employed arsenic in the treatment of skin diseases, states that when the susceptibility is not great, a dose of *two drachms* of the solution (= *one grain* of arsenic) can be borne about as well in *one* dose as in twenty. He quotes a case in which a patient took *two drachms* of the solution. (= one grain of arsenic) in twenty-four hours, by mistake. It cured the ague, for which it was prescribed, and had no injurious effect. (*Med. Times*, Sept. 14, 1850, p. 270.) It is difficult to explain such anomalies by varying susceptibility only; they are more probably due to the uncertainty of composition in the preparation employed.

The *treatment* of a case of poisoning with a soluble akaline arsenite, would be the same as that for arsenious acid; but the hydrated oxide of iron and magnesia might be given with a greater prospect of benefit.

Analysis.—This solution has the odor of tincture of lavender, is of a reddish color, and has an alkaline reaction. It gives at once a green precipitate (arsenite of copper) with the sulphate of copper, and a yellow precipitate with nitrate of silver. Acidulated with hydrochloric acid, and treated with a current of sulphuretted hydrogen gas, it yields a yellow sulphide; and when boiled with hydrochloric acid and copper, a metallic deposit is obtained which readily furnishes octahedral crystals of arsenious acid. If one drop (= 120th grain of arsenic) is added to a concentrated and boiling solution of chloride of tin mixed with fuming hydrochloric acid, the arsenic is reduced and deposited as a dark brown or black precipitate.

ARSENITE OF SODA.

The *arsenite of soda* is as poisonous as the arsenite of potash. In December, 1857, three hundred and forty children belonging to an industrial school near London were poisoned by this compound mixed with arseniate. It had been incautiously used for cleansing a steam-boiler, and had thus been mixed with the hot water which was drawn for the breakfasts of the children. The dose of arsenic taken by each child I found to be about one grain. All recovered, although some suffered severely. (For an account of the symptoms in this large number of cases, the reader is referred to a preceding page, 290.) In the winter of 1863, a man died under symptoms of acute poisoning by arsenic, owing to his having drunk beer out of a pot which had contained this *patent* cleansing fluid.

Analysis.—The presence of arsenious acid in this compound may be determined by all the usual tests. Bettendorff's test, the acid chloride of tin, gives at the boiling-point a large deposit of arsenic from a small quantity of the solution.

ARSENITE OF COPPER—SCHEELE'S GREEN—EMERALD GREEN.

The poisonous properties of this compound are undoubtedly due to the arsenic which it contains; hence it may be appropriately considered with the arsenites. It is the only metallic arsenite which is met with in commerce and the arts, and it constitutes, wholly or in part, a great variety of green pigments, known as Emerald green (aceto-arsenite of copper), mineral green, Brunswick, Schweinfurt or Vienna green, etc. It is thus found in the form of oil-paint, in cakes, in boxes of water colors, spread over confectionery, in wafers, in adhesive envelopes, and lastly, and most abundantly, in the various kinds of green decorative papers for covering the walls of sitting and bed-rooms. A London manufacturer informed me that at one time so great was the demand for this "cheerful" but poisonous color, that his average consumption of arsenic had amounted to about two tons weekly! The extensive diffusion of arsenic in this pigment, with the facility which it gave for universal poisoning when the knowledge and inclination coexisted, was actually converted into an argument to show the inutility of placing any legal restrictions on the sale of arsenic or other poisons! The German government, after instituting inquiries, had solved the question, by simply prohibiting the manufacture, sale, and use of these arsenical papers. In this country, however, the arsenical green decorative papers are unconsciously purchased by the public, and are most extensively used in dwelling-houses, especially among the poorer classes, by reason of the cheapness and durability of the green colors! We may first consider the effects of Scheele's green in its ordinary state of powder.

Symptoms and Effects.—Although the arsenite of copper is insoluble in water, it is sufficiently soluble in the acid mucous fluids of the stomach to be taken up by the absorbents, and carried as a poison into the blood.

In several cases in which children have eaten portions of a green water-color pigment containing arsenite of copper, severe pain, with vomiting, purging, cold sweats, and intense thirst, were among the symptoms. These symptoms gradually subsided, and they recovered. (*Med. Times*, 1849, p. 507; *Guy's Hospital Reports*, October, 1850, p. 218; *Med. Gaz.*, vol. 43, p. 304; *Ed. Monthly Jour.*, July, 1851, p. 1; and *Lancet*, March 5, 1859, p. 237.) In two cases referred to me in January, 1853, the children died from eating a portion of a confectionery ornament picked up in the street. It was a layer of sugar and starch, colored with the arsenite of copper. M. Chevallier has lately published other cases of a similar kind. (*Ann. d'Hyg.*, 1874, vol. 1, p. 92.)

The late Dr. Geoghegan informed me that an accident occurred in Dublin in 1842, by which fourteen children suffered from symptoms of poisoning, in consequence of their having eaten some confectionery ornaments colored with arsenite of copper. In two or three of the cases jaundice followed. The dangerous practice of using this powerful poison to give a color to confectionery was at one time very prevalent, but it is not now so frequent. An instance was communicated to me in which three lives were nearly sacrificed at a school near Manchester, owing to the boys having eaten some ornamented confectionery, which owed its green color to arsenite of copper. They suffered from violent vomiting, severe pains in the stomach and bowels, and spasms in the arms and legs. Three animals which ate a portion of the vomited matters were attacked with similar symptoms.

In a case which was the subject of a criminal trial, this compound was proved to have caused the death of a gentleman by reason of it having been employed to give a rich green color to some blancmange served at a public dinner—the person who employed it considering that emerald or mineral green was nothing more than an *extract of spinach!* It led to death under the usual symptoms, and the parties were convicted of manslaughter, and sentenced to imprisonment. (*Reg.* v. *Franklin and Randall*, Northampton Summer Assizes, 1848.) Most of the colors used for confectionery are of a poisonous nature; the pink color given by cochineal or madder is the only one which can be regarded as innocent.

Arsenicated Wall-papers.—One of the uses to which this noxious compound is put is the manufacture of wall-papers.

The pigment contains fifty-nine per cent. of arsenic, and from some of these green papers in the unglazed state, the noxious material may be easily scraped or removed by slight friction. A square foot may yield from fourteen to seventy grains of the arsenical compound, and in rooms exposing five or six hundred square feet, arsenic is liable to be distributed through the air of a room in the state of a fine dust or powder. I have detected this poisonous dust on books, picture-frames, furniture, and projecting cornices in rooms thus papered. Workmen who hang these papers, or who strip them off the walls, are well known to suffer from symptoms referable only to the action of arsenic. (See Husemann, *Jahresbericht der Toxicologie*, 1871, p. 425, and *Pharm. Jour.*, 1870, p. 218; also *Lancet*, 1870, vol. 2, p. 356.) One of my friends who had his library papered with an arsenicated wall-paper, suffered from symptoms of arsenical poisoning, which came on after he had been occupied in dusting his books. I examined the dust, and found therein a well-marked quantity of arsenic.

Dr. Böcker, of Bonn, a recent writer on Toxicology, refers to the effects of chronic poisoning produced in persons inhabiting rooms of which the walls are covered with arsenical paperhangings, and states that on several occasions he has been called upon to treat such cases. A removal of the cause has generally proved sufficient. Dr. Böcker considers that a damp state of the wall renders them especially injurious. (*Die Vergiftungen*, 1857, p. 132; Casper's *Vierteljahrs.*, 1858, p. 181.)

Dr. G. Kirchgässer, of Coblenz, has published an elaborate paper on this chronic form of poisoning, which he calls *arsenicism*. He has collected twenty-one cases of poisoning, as the result of persons inhabiting rooms the walls of which were covered with a green arsenical pigment. Some of these, he states, proved fatal, and arsenic was in some cases detected in the urine of the patients. (Horn's *Vierteljahrsschrift*, 1868, vol. 2, p. 96; also *Annales d'Hygiène*, 1869, vol. 1, p. 480.) It is probable that if sought for, it would also be found in the saliva; this might aid diagnosis. M. Delpech has published some facts which show that similar symptoms of poisoning have arisen from a person occupying a room filled with stuffed birds and animals, in the preservation of which an arsenical compound had been used. Arsenic was found in the dust of the room and on the furniture. (*Ann. d'Hyg.*, 1870, vol. 4, p. 314.)

The *symptoms* produced by arsenicated wall-papers are of a uniform character, showing their origin from a common cause. They are as follows: dryness and irritation of the throat with cough, irritation of the mucous membrane of the eyes and nostrils, dry cough, shortness of breathing, languor, headache, loss of appetite, nausea, colicky pains, numbness, cramp, irritability of the bowels attended with mucous discharges, great prostration of strength, sleeplessness, a feverish condition, and wasting of the body. Those symptoms may not all present themselves in any one case; they are derived from the examination of numerous cases which have been referred to me. They resemble those of a severe cold. No suspicion of the real cause has been entertained until after all ordinary treatment had failed to impart relief, and an analysis of the paper had been made. The connection of the symptoms with this cause appears to have been in some instances clearly established by the fact that after the removal of the paper, especially from bed-rooms, the symptoms have disappeared. (*Med. Times and Gaz.*, vol. 1, p. 647.) It is, however, proper to observe that, as in reference to the manufacture of white lead, comparatively few of those who are exposed, suffer from symptoms of poisoning.

It is probable that to the noxious practice of covering the walls of our sitting- and bed-rooms with arsenic, many insidious cases of illness and chronic disease may he referred. It is not only by the dust that the poison is disseminated, but according to the recent experiments of Dr. Hamberg, a decomposition takes place, by which the poisonous gas, arseniuretted hydrogen, is produced and diffused through the air. By a properly arranged apparatus, he was enabled to collect this gas in a solution of nitrate of silver, and to establish its presence in the air in the room by the process described at p. 321, *ante*. (See a paper by Dr. Hamberg, of Stockholm, in *Pharm. Journal*, August, 1874, p. 82.) Dr. Hamberg's results are in strict accordance with the physiological facts, and we now learn conclusively that those who occupy arsenicated rooms are exposed to the risk of breathing a poisonous gas of the most deadly kind. Some persons may have a tolerance for arsenic and resist the effects, but there are others who by idiosyncrasy are less tolerant of this poison, and who are likely to suffer. It is now placed beyond doubt that there are two sources of poisoning under these circumstances: 1, the impalpable dust which is diffused through the room; and, 2, the noxious gas produced by the slow decomposition of the aceto-arsenite of copper. For further observations on the effects of these arsenical pigments on health, I must refer the reader to Dr. Guy's *Report to the Privy Council*, 1862, p. 126; to papers by Dr. Verner (*Ann. d'Hyg.*, 1859, vol. 2, p. 346), and by Dr. G. Johnson (*Sanitary Record*, Nos. 1 and 2, July, 1874).

This arsenical compound is much used for coloring artificial flowers, wreaths, and tarlatan dresses. Girls employed in this manufacture, as well as dressmakers, suffer seriously from this form of poisoning. Two women were employed to make some green tarlatan into ball-dresses. They noticed an unpleasant smell and taste, and their eyes were affected during the performance of the work. The symptoms from which they suffered were swelling of the eyelids, congestion of the conjunctivæ, and copious secretion of tears. The one most affected experienced on the second day salivation, with an unpleasant taste in her mouth, cramps in the limbs, great thirst, restlessness, and difficulty of breathing. These symptoms lasted in one patient eight, and in the other, fourteen days. Rieder, a chemist of Berlin, who describes these cases, suffered severely from similar symptoms for several days, as a result of handling the poisoned dresses for the purpose of analysis. He found that the stuff contained thirteen per cent. of its weight of arsenic. (Husemann, *Jahresbericht der Tox.*, 1871, p. 525; also *Jahresbericht*, 1872, p. 480.) The color is loosely laid on with starch, and it comes off on the slightest friction in a cloud of poisonous dust. Dr. G. Johnson states that a dress of this kind contained half its weight of the arsenical green. Three grains and a quarter of arsenic were separated from a square foot of the green gauze (*Sanitary Record*, July 11, 1874). I may add to this list the case of a lady (July, 1872), who suffered from symptoms of arsenical poisoning, by reason of her having worn, on one occasion only, a dress of this description. Paper used for adhesive envelopes, for wrapping confectionery, children's food, isinglass, chocolate, etc., is also frequently colored with it. Under proper sanitary legislation the manufacture and sale of this paper would be prohibited. It has been stated that the green woollen of carpets owes its color to this compound, but I have not found it in any English samples nor in the green Berlin wool.

Analysis.—For the chemical characters of SCHEELE'S GREEN, see p. 309. The wall-pigment called EMERALD GREEN is a mixture of arsenite and acetate of copper. The color is, even by candlelight, a vivid green, but it is sometimes diluted with chalk to a very pale sea-green. The presence of arsenic in the compound can be easily detected by all the tests for arsenic (page 306); but the following is a simple method, which admits of speedy application. A slip of the suspected paper should be soaked in a moderately strong solution of ammonia. The green color is removed, and the blue ammoniuret of copper is formed and dissolved in a few minutes. This result establishes only the presence of a compound of copper soluble in ammonia. If the ammonia does not become blue, there is no arsenite or copper-salt present; if it does become blue, a crystal of nitrate of silver should be placed in a white saucer and a small portion of the blue liquid poured over it. The presence of arsenic is revealed by the production of yellow arsenite of silver over the surface of the crystal (p. 309). Another method consists in adding a fragment of the paper to boiling chloride of tin, acidulated with fuming hydrochloric acid. Metallic arsenic is precipitated of a dark brown color. A small portion of the paper dissolved in hydrochloric acid added to the apparatus represented in Fig. 25, p. 323, will set free arsenic in the form of arseniuretted hydrogen.

When the green pigment is used in confectionery, the substance upon which it is spread is either soluble (sugar or starch) or insoluble (plaster of Paris). In either case we scrape off the green color and digest it in a small quantity of water. In the first case the arsenite of copper is deposited, while the sugar or starch is dissolved; in the second, the arsenite of copper is deposited with the sulphate of lime. The former may be separated from the latter by ammonia, and reobtained pure by evaporation. Should the arsenite be mixed with fat or oil, it will easily subside as a sediment on keeping the substance melted, and the deposit may be freed from any traces of fat by digesting it in ether or sulphide of carbon. The presence of arsenic in the compound may be easily detected by the processes above described.

CHAPTER XXXVII.

METALLIC ARSENIC—FLY-POWDER—ARSENIC AND ALKALINE ARSENIATES—ANILIN COMPOUNDS CONTAINING ARSENIC—THEIR EFFECTS—SULPHIDES OF ARSENIC—ORPIMENT—CHLORIDE OF ARSENIC—ARSENIURETTED HYDROGEN.

METALLIC ARSENIC—FLY-POWDER.

It is generally considered that metallic arsenic is not poisonous, but as this metal is easily oxidized it speedily acquires poisonous properties. According to Berzelius, the metal is slowly converted, by exposure to the air, to a pulverulent suboxide of a black or brownish-black color. This is commonly called "fly-powder," a name also applied to the arsenical cobalt ores reduced to powder. Thus, what is called the "Tunaberg ore"—a mixture of cobalt, arsenic, iron and sulphur—is largely used on the continent under the name of "fly-powder," and as it comes within the reach of children it frequently gives rise to accidents.

Symptoms and Appearances.—A few years ago, Dr. Schobbens was called to a man who had taken some of this powder by mistake for a purgative. He was soon attacked with the usual symptoms of poisoning with arsenic. He swallowed a large quantity of milk, which occasioned immediate vomiting. As fifteen hours had elapsed before a medical man saw him, no treatment was of any avail, and he died from the effects of the poison. In another case, a child, aged four years, swallowed a portion of fly-powder. The hydrated sesquioxide of iron was given every half-hour, and the child recovered the next day. (*Monthly Jour. Med. Science*, Sept. 1846, p. 228.) The exact quantity taken in this case is not known, but there is no doubt that the poison is but little inferior to arsenious acid in activity, and the symptoms and appearances after death from a fatal dose would be similar. This substance is not much known in England. A woman was convicted in France of poisoning her husband with it in 1844. (Briand, *Man. Comp. de Méd. Leg.*, p. 228.) It owes its poisonous properties to arsenious acid, of which, with the metal, it appears to be a mechanical mixture.

According to Dr. Schütte, it contains, as it is usually sold, from four to eleven per cent. of white arsenic. This gentleman has reported one case in which it was homicidally administered by a man to his wife. The prisoner, Dombrowsky, was tried before the Court of Wolfenbuttel, in July, 1853, convicted of murder by poison, and subsequently executed. The quantity of the powder administered to the deceased is unknown. The symptoms from which the woman suffered were violent vomiting and purging, severe pain in the abdomen, and great thirst. She died in six days. The principal appearances were softening and excoriation of the mucous membrane of the stomach, striped or striated inflammatory redness of the mucous membrane, with bloody points about the cardia. The intestines were also inflamed. Small, black, metallic-looking particles were found in the contents of the stomach; and these, on analysis, proved to be arsenic. The quantity found amounted to about nineteen grains. Some of the same kind of powder was taken from the prisoner's pocket. (Casper's *Vierteljahrsschrift*, 1854, vol. 2, p. 230; and Otto, *Ausmit. der Gifte*, 1856, p. 56.)

This metallic-looking powder forms what may be called *black* arsenic. Dr. Chevers refers to a case that occurred in India, in which some difficulty arose in respect to the evidence, in consequence of a witness describing an arsenical powder as black. (*Med. Jur. for India*, p. 74.) It was thought that the witness had made a mistake in the description, but the arsenic was probably in the form of fly-powder. It would be known from white arsenic colored by soot, by the great weight and metallic appearance of the black particles.

Analysis.—When boiled in water, arsenious acid is dissolved, and the appropriate tests may be then applied to the solution. When a small portion is gently heated in a reduction-tube, a ring of arsenious acid is obtained, as well as a ring of metallic arsenic. With soda-flux, a well-defined metallic crust is procured, possessing the characters already described (*ante*, p. 306). This compound is used for destroying flies as well as vermin.

Fly-water is a name applied to a solution of arsenic and of various arsenical compounds in water. Mixtures of this kind may be formed by dissolving one part of the arsenite of soda or potash, and two parts of sugar, in twenty parts of water. Paper soaked in this solution, and dried, is used for poisoning flies; and perhaps this is the safest form in which arsenic can be used for such a purpose. A case of poisoning by fly-water, in which two grains and a half of arsenious acid destroyed the life of an adult in thirty-six hours, is reported in the *Medical Gazette* (vol. 39, p. 116).

ARSENIC ACID.

Symptoms.—This is an artificial product almost entirely confined to the chemical laboratory. Orfila states that it is a more powerful poison than arsenious acid, but he does not adduce any cases in support of this opinion. I have not been able to find any instance of poisoning by it in the human subject. Dr. Glover ascertained that four grains of the acid, dissolved in two drachms of water, and introduced into the stomach of a stout rabbit, killed the animal in four hours, with the symptoms of irritant poisoning, and an affection of the nervous system. (*Ed. Med. and Sur. Jour.*, vol. 58, p. 121.)

Treatment.—The hydrated oxide or acetate of iron would be more likely to act as an antidote in poisoning with arsenic acid, owing to the great solubility of this compound and its tendency to combine with the oxide.

Analysis.—Arsenic acid is a white uncrystalline deliquescent solid. 1. It is not entirely volatilized on platinum-foil by the flame of a lamp. 2. It is very soluble in water, forming a highly acid solution. 3. It is precipitated of a brick-red color by nitrate or the ammonio-nitrate of silver. In these characters it differs from arsenious acid. The smallest quantity may be detected by this test (see *ante*, p. 307.) 4. It yields readily an arsenical sublimate with charcoal. 5. It yields deposits with copper and hydrochloric acid, as well as in Marsh's apparatus. Dr. Rainey has shown that Reinsch's process does not act so delicately with the arsenic, as with arsenious acid. The arsenic may, however, be converted into arsenious acid by mixing it with sulphurous acid and evaporating the liquid to dryness. Arsenic acid is also precipitated, but of a pale yellow color, and slowly, by sulphuretted hydrogen gas. In some of these properties it resembles arsenious acid. There are two compounds of arsenic in the British Pharmacopœia,—the arseniate of soda and the arseniate of iron.

ARSENIATES OF POTASH AND SODA.

Symptoms and Appearances.—The arseniates of potash and soda must be regarded as active poisons, although there are but few instances on record in which life has been destroyed by them. Sir R. Christison states that, in the course of his reading, he has met with only two reported cases of poisoning by arseniate of potash. (*Op. cit.*, p. 284.) M. Bouley administered this salt to seven horses, from the effects of which they all died. On inspection, it was observed that there was well-marked inflammation of the stomach, intestines, and bladder, and there were ecchymoses in the left ventricle of the heart. The contents of the viscera in one horse yielded no traces of arsenic—a fact probably to be ascribed to the violent purging from which the animal had suffered. (Orfila, *Toxicologie*, vol. 1, p. 452.)

An attempt at murder by the arseniate of potash was the subject of a trial in France in 1844. This poisonous salt had been maliciously put into a bottle of wine. The prosecutor swallowed a mouthful of the wine, and from finding it very bitter, he spat out the greater portion. His wife also tasted it, but drank only a small quantity. In the course of the night the prosecutor was seized with severe colic, vomiting, great prostration of strength, and stupor; the wife suffered from similar symptoms. The medical man who had been called to them, finding that but a small portion of the wine had been taken from the bottle, referred the symptoms to indigestion. The next morning the prosecutor gave a portion of the suspected wine to a dog: the animal suffered from violent vomiting and convulsions, and died in four hours. The wine was analyzed by M. Chevallier, and found to contain about one drachm of arseniate of potash to a pint. A person, in whose possession a large quantity of arseniate of potash was found, was tried for this nefarious attempt to poison; but he was acquitted. (*Journal de Chimie Médicale*, 1854, p. 254.) A coarse sort of bibulous paper, soaked in a solution of arseniate of potash, is now extensively sold under the name of "*Papier Moure*." It is erroneously represented that the substance with which it is impregnated is not poisonous to human beings. (*Lancet*, Feb. 11th, 1860; also *Annales d'Hygiène*, 1860, vol. 1, p. 292.)

Two cases are reported of poisoning by arseniate of soda. Two young men sent to a druggist's for doses of tartrate of soda, in place of which arseniate of soda was sent by mistake. In five minutes after the substance had been taken, they were attacked with cramps in the stomach. One died, and the other remained for some time in a dangerous condition. (*Amer. Jour. Med. Sci.*, Oct. 1852, p. 553; and Wharton and Stillé's *Med. Jur.*, p. 454.)

Arsenic acid is largely employed in the manufacture of magenta, rosanilin, and other colors from anilin. There is reason to believe that the color is often sent into the market contaminated with arsenic. Dr. Rieckher has found from one to seven per cent. of arsenic acid in the red colors supplied by good manufacturers, and frequently arsenious acid was also present. (*Med. Times and Gaz.*, 1870, vol. 1, p. 617.) As these compounds are used for giving a "beautiful" red color to liqueurs, syrups, raspberry-vinegar, and sugar-sweetmeats, there is a possibility that accidents may occur from their use. They supplant all others by reason of their richness of tint and great cheapness. (*Med. Times and Gaz.*, 1870, vol. 1, pp. 46, 84.) From the facts collected by M. Charnet, it appears that those who are engaged in this manufacture suffer from cutaneous eruptions of a pustular kind, with œdema of the skin, colic, diarrhœa, vomiting, salivation, paralysis, and other symptoms showing an affection of the nervous system. The cutaneous eruptions are similar to those observed in workmen engaged in the manufacture of green arsenical papers. (*Ann. d'Hygiène*, 1863, vol. 2, p. 281.) In the factory which he examined, arsenic acid was largely employed in the conversion of anilin into *fuchsin*, and the arsenic was manufactured on the premises, by the usual process of mixing arsenious acid with aqua regia. In spite of all precautions, he found that arsenical compounds were diffused through the air in small proportion. Traces of arsenic were found in the purest anilin red (fuchsin), in the dust of the factory on the floor, and in the air breathed by the workmen! Those who suffered had been exposed to their influence one or two weeks before symptoms occurred. These sometimes assumed an acute, and, in other cases, a chronic form.

Anilin red, or fuchsin, is soluble in water, and is recognized by the red color being completely discharged when a solution of ammonia is added to it, and by the restoration of the red color on the addition of an acid. The presence of arsenic acid in it may be detected by Marsh's process, or by any of the methods already described. Even when the color does not contain arsenic, the mordant used for fixing it may be the source of poison. Dr. Bijon found that he suffered from repeated attacks of inflammation of the eyes when he slept in a room of which the walls were covered with an unglazed *red* paper. The red dye on the paper was coralline, a color derived from carbolic acid. It contains no arsenic, but, according to Tardieu and Roussin, it is itself a powerful poison—(*Ann. d'Hyg.*, 1869, vol. 1, p. 267)—although this is disputed by Bouchardat. (*Ann. d'Hyg.*, 1874, vol. 2, p. 170.) The symptoms suffered by Dr. Bijon were similar to those caused by the arsenical green papers, and further experiments showed that a mordant of arseniate of alumina has been employed to fix the coralline red to the paper. It is obvious, therefore, that the symptoms of poisoning may be produced with paper of any color, if the color is fixed by an arsenical mordant. M. Bouchardat very properly directs the attention of the authorities to this new introduction of poison. He states that one of his assistants, who was cutting up for analysis, the coralline paper supplied by Dr. Bijon, suffered from great irritation of the eyelids. (*Ann. d'Hyg.*, 1874, vol. 2, p. 173.)

It has been lately announced, on good chemical authority, that certain manufacturers of printed fabrics have substituted for albumen the arseniate of alumina and a compound of arsenic acid and glycerin. In one yard of stuff there may be as much as from thirty to forty grains of arseniate of alumina. This has been especially observed in printed calicoes with a yellowish-brown or brownish-red pattern, shades hitherto unsuspected, which the uninitiated buy without suspecting the danger incurred in wearing them. The arsenic is not in an insoluble form, for on the fabrics being placed in water for a few seconds they give off a considerable quantity of it. These productions are generally sold at a low price, and they have evidently not been washed after being printed, as the water would have removed a portion of the color.

Treatment.—When arsenic acid or an arseniate has been taken as a poison, hydrated oxide of iron may be freely given. Owing to the great solubility of these compounds, the iron antidote may be administered in these cases with a fair prospect of benefit.

Analysis.—Arseniate of potash is a white deliquescent substance, fixed when heated, and very soluble in water. The same tests may be applied to it as to ARSENIC ACID. Marsh's process acts with much more certainty and delicacy than that of Reinsch (*ante*, p. 311). In order to separate the whole of the arsenic, the liquid may be acidulated with hydrochloric acid, and a current of sulphuretted hydrogen gas passed into it.

The BINARSENIATE OF POTASH is known under the name of *Macquer's neutral arsenical salt.* The liquid known as Pearson's solution, which is still used medicinally in France, is a mixture of one grain of *arseniate of soda* to one ounce of distilled water.

SULPHIDES OF ARSENIC.

There are several kinds met with in commerce—ORPIMENT or YELLOW ARSENIC, and REALGAR or RED ARSENIC. They are poisonous in consequence of their containing a large proportion of free arsenious acid; this sometimes amounts to as much as 30 per cent. of their weight. They are occasionally used as poisons; in several criminal cases in England it has been proved that orpiment was the substance employed. Orpiment (*Auri pigmentum*) from its rich golden yellow color, and realgar are employed in the arts, and are procurable by artisans with the most destructive facility. On one occasion, a quantity of red powder, brought to me by a mechanic as iron rust, which he was carrying loosely in his waistcoat pocket, turned out to be realgar! From the brilliant colors of these compounds, they are used in painting, dyeing, and even in the coloring of toys and sweetmeats for children! It is remarkable that, under these circumstances, accidents are not more frequent. (See *Ann. d'Hyg.*, 1843, vol. 1, p. 358.)

In December, 1859, six persons suffered from the usual symptoms of poisoning with arsenic, owing to their having eaten *Bath buns.* It was found that a confectioner at Clifton had used, as he supposed, chromate of lead to give the buns a rich yellow color, and make them salable; but the druggist to whom he applied had ignorantly supplied him with orpiment. This wholesale system of poisoning is one of the attendant evils of adulterating articles of food. The Bradford lozenge case (Nov. 1858) furnishes a remarkable instance of the impunity attendant upon acts of this kind. A confectioner, intending to adulterate lozenges with plaster of Paris, mixed with them a quantity of white arsenic which had been supplied to him through mistake. I am informed that more than 200 persons partook of those poisoned lozenges, and suffered the usual effects. Seventeen persons died; twelve from acute poisoning, and five from the secondary effects. A trial took place, but the law could not fix the responsibility for this act upon any person.

It is in the state of yellow sulphide that arsenic is so commonly found in the stomach after death, when the body has been buried for a long period. This arises from the action of sulphuretted hydrogen, generated by decomposition, on the white arsenic taken during life. In some instances the coats of the stomach and intestines, the liver, diaphragm, and even the bones of the spinal column, may be thus deeply stained of a yellow color.

Symptoms and Appearances.—The sulphides of arsenic produce symptoms and appearances similar to those caused by arsenious acid; but the dose required to destroy life will vary according to the proportion of arsenious acid with which the sulphide happens to be mixed. This is not a common form of poisoning; the intense color given by the poison to food would generally excite suspicion. It was with orpiment that Mrs. Smith was poisoned at Bristol in 1835. (*Med. Quart. Rev.*, July, 1835, p. 390.) This poison, owing to its color, may be given or taken, by mistake, for mustard or turmeric. In a case which occurred to Dr. Jochner, two persons partook of some porridge, in which orpiment had been put by mistake for turmeric. They suffered from continual vomiting, burning pain in the stomach, and collapse. One, an old man, died in twenty-two hours; the other, a boy, recovered. Evidence of violent inflammation was found in the gullet and stomach, the mucous coat of the latter being thickened. There was a sphacelated spot, one inch in diameter, in the œsophagus; and another in the stomach, three inches in extent. (Wharton and Stille, *Med. Jur.*, p. 434.) According to Dr. Chevers (*Med. Jur. for India*, p. 74), orpiment is much used in India both as a medicine and as a poison. He refers to eight instances in which this poison was found, either in food or in the stomachs of persons who had died under symptoms of irritant poisoning. The sulphide was detected in the remains of two bodies, after ten months' interment without coffins. Orpiment and realgar are sold openly in India, and are used as depilatories. Orpiment has been known to cause death by *external* application as a depilatory (see *Ann. d'Hygiène*, 1834, vol. 1, p. 459); a result which might be expected from the quantity of arsenious acid with which it is mixed. There is a form of depilatory used, which consists of one part of orpiment, twelve parts of quicklime, and ten parts of starch, made into a soft paste with water (*Pereira*, vol. 1, p. 162), the application of which to the skin must always be attended with danger.

Another compound, containing sulphide of arsenic, mixed with lime and sulphur, is extensively sold as a pigment, under the name of *King's* or *Naples yellow*. It is of a pale yellow or lemon color. Dr. Paterson has reported the following case of poisoning with it; but it will be seen that the cause of death was not clearly traced to this substance: A girl, æt. 18, swallowed a drachm of King's yellow. In about two hours she began to vomit, and she still vomited when admitted into the Edinburgh Infirmary, *i. e.*, about ten or twelve hours after having taken the poison. When admitted there was great anxiety, with collapse, coldness of the surface, and a scarcely perceptible pulse. On the day following the signs of irritation in the alimentary canal abated under treatment, and symptoms of acute bronchitis supervened. She died sixty hours after having taken the poison. Appearances indicative of inflammation were found in the air-passages and lungs; but there was no sign of active inflammation in the stomach, and the intestines were healthy throughout. There could be no doubt that the immediate cause of death was bronchitis; but the question to be decided was, whether this had arisen from the usual accidental causes, or whether it had been produced by the poison. From the fact that the mucous membrane of the air-passages has been occasionally found inflamed, in cases of arsenical poisoning, Dr. Paterson concluded that the arsenic was here the remote cause, and that the inflammatory action probably extended by continuity from the alimentary canal into the air-passages. (*Monthly Jour. of Med. Science*, Sept. 1846, p. 184.)

In *Reg.* v. *Sturt* (Lewes Lent Assizes, 1863), a novel chemical question arose respecting the sulphide of arsenic. There was some reason to believe that the deceased woman had died from the effects of arsenic administered in confectionery. White arsenic was found in the stomach, and a question was put by the learned judge, as well as by the counsel for the prisoner, whether the confectioner might not have used yellow arsenic by mistake in order to color the substance, and this yellow arsenic have been converted in the deceased's body, in twenty-four hours, into white! It need hardly be remarked that the yellow color is an essential character of orpiment. White may be converted into yellow arsenic in the dead body, but yellow cannot be spontaneously changed into white arsenic.

Treatment.—The promotion of vomiting with the exhibition of mucilaginous liquids, and the use of the stomach-pump, can alone be trusted to.

Analysis.—The powdered sulphides yield a solution of arsenious acid on boiling them in water acidulated with hydrochloric acid. They readily give the well-known sublimates of metallic arsenic, with the cyanide of potassium flux, and metallic deposits with the hydrogen apparatus. They also yield a deposit of arsenic when boiled with copper and hydrochloric acid.

The presence of arsenious acid in common orpiment may be also determined by boiling it with chloride of tin and hydrochloric acid. The arsenic is immediately precipitated in the form of brown deposit. The pure sulphide, produced by the action of sulphuretted hydrogen on a solution of arsenious acid, does not undergo this change.

Common orpiment may be distinguished in *organic mixtures* by its color, and separated as a sediment by its great density. As it always contains arsenious acid, chloride of arsenic may be obtained from it by distillation (see *ante*, p. 320).

This remark equally applies to the yellow arsenic found in the stomach or intestines after death as the result of the conversion of the poison. When dried and distilled with fuming hydrochloric acid, chloride of arsenic is obtained. The sulphide free from arsenious acid yields no chloride under these circumstances.

HYDROCHLORIC SOLUTION OF ARSENIC—(LIQUOR ARSENICI HYDROCHLORICUS).

This is a pharmacopœial solution of arsenic in diluted hydrochloric acid. It contains four grains of arsenious acid in one fluid ounce, and is of the same strength as liquor arsenicalis. Mr. Phillips describes it as a highly poisonous preparation, and from a case which I saw in Guy's Hospital in May, 1857, this statement is correct. A woman took, in three doses, thirty minims of the old preparation over a period of twenty-four hours. The quantity of arsenic thus taken was not more than the *tenth part* of a *grain*, and yet the symptoms which followed were of a severe kind, resembling those of chronic poisoning. There were constriction of the throat, pain and irritation of the stomach and bowels, tingling and numbness of the hands and feet, loss of muscular power, and a feeling of extreme depression. The medicine was withdrawn, and the patient slowly recovered. It seems that the woman had not taken arsenic previously, and there was no evidence of the existence of a peculiar susceptibility to the effects of this poison. The quantity taken was very small to have produced such alarming effects. The medicinal dose of the new solution is from two to eight minims.

Analysis.—This compound is obtained in the separation of arsenic from organic solids by distillation (see p. 320). It may be tested by the process of Marsh or Reinsch's, as there described. When boiled with fuming chloride of tin it is decomposed, and metallic arsenic of a brown-black color is precipitated. Chlorine may be detected in it by nitrate of silver.

ARSENIURETTED HYDROGEN.

This is a gaseous poison of arsenic, producing, when breathed even in small quantity, very serious effects upon the system. It has already occasioned death in at least six instances. (*Chem. News*, Dec. 26, 1863, p. 307, and *Jahresbericht der Toxicologie,* 1871, p. 522.) The gas is an artificial product, and is formed in a chemical laboratory in various ways—one method has already been described in speaking of Marsh's process (*ante*, p. 311); and its highly poisonous properties render it necessary that caution should be used in the employment of this mode of testing. The gas is most effectually decomposed, and prevented from diffusing itself, by passing it into a solution of nitrate of silver, or chloride of gold.

This form of gaseous arsenical poisoning has been hitherto purely accidental. It is stated that Gehlen, a German chemist, was killed by accidentally breathing a small quantity. Suspecting that the gas was escaping from some part of the apparatus which he was using, he applied his nose for the purpose of detecting it; and although he respired but a small quantity, probably a few hundredths of a grain of arsenic only, he was seized in about an hour afterwards with vomiting, shivering, and great prostration of strength. He died on the ninth day. The most complete history of this kind of poisoning has been published by Dr. O'Reilly, of Dublin. I am indebted to him for the particulars of the case.

Symptoms and Appearances.—A gentleman, for the sake of experiment, wished to respire about one hundred and fifty cubic inches of pure hydrogen gas. It unfortunately happened that the sulphuric acid, which he used for making the hydrogen, was largely contaminated with arsenic. Immediately after he had respired the gas, he was seized with giddiness and fainting, constant vomiting of a greenish-colored matter, and a dull pain in the region of the stomach. There was also complete suppression of urine. He died in about six days. On dissection, the liver and kidneys were found of a deep indigo color; the mucous membrane of the stomach was easily separated, and there were two distinct patches of inflammation at the greater curvature. There was a quantity of reddish-colored fluid effused in the chest, in about ten ounces of which Dr. O'Reilly detected arsenic by the use of Marsh's process. From experiments made subsequently on the sulphuric acid, it is supposed that the deceased must have inhaled a quantity of arsenic equivalent to about twelve grains of arsenious acid. Another case of poisoning by this gas occurred in England in December, 1836. A young chemist was killed by respiring the gas, evolved from a mixture of arsenic, zinc, and sulphuric acid. Death did not take place until twenty-four days after the accident. It appears that in this instance but a very small portion could have entered into the lungs. I am indebted to Dr. Mouatt, of Calcutta, for the particulars of a third case, that of Prof. Robertson, of Calcutta Medical College, who, while delivering a lecture on arsenic, accidentally breathed a portion of this gas which was escaping from a Marsh's apparatus. The first symptoms were a sense of burning and of constriction in the throat, followed by irritability of the stomach, vomiting of liquid, at first bilious and afterwards coffee-colored, with a burning pain through the whole alimentary canal. Four pints of bloody urine were passed, and this, on examination, was found to contain arsenic. There was constipation of the bowels, with fever, a full, hard, frequent pulse, dry, hot, unperspiring skin, restlessness, anxiety, and great prostration of strength. He did not recover from these symptoms until the twenty-second day.

Other chemists who have incautiously breathed the deadly vapor while performing scientific experiments have also suffered from similar symptoms. Dr. Frost, of Aachen, has lately reported three fatal cases, with a minute account of the symptoms and appearances. These occurred accidentally among workmen engaged in separating silver from lead by means of zinc and hydrochloric acid. The latter was found to contain much arsenic, which escaped with the hydrogen, and caused the deaths of the workmen. (*Vierteljahrs.*, 1873, vol. 1, p. 269.)

Analysis.—The chemical properties of this gas have been already described. (See MARSH'S PROCESS, *ante*, p. 311.) It is colorless, possessed of a disagreeable odor resembling that of garlic, inflammable, burning with a bluish-white flame, and evolving an abundance of white solid vapor. While burning, it is converted into water and arsenious acid. On cold surfaces it deposits metallic arsenic, suboxide of arsenic, arsenious acid, and water. It is decomposed by chlorine, forming hydrochloric acid and chloride of arsenic; also by those metallic solutions the metals of which have a weak affinity for oxygen. Paper soaked in a solution of nitrate of silver and held over the gas, is immediately blackened. If the gas is passed into the solution, the silver is reduced, and arsenious acid is dissolved. The gas is not soluble in water, and the air of water decomposes it. At a red heat the metal is entirely deposited on the interior of the glass tube, and hydrogen escapes. This is applied as an adjunct test in Marsh's process. It is known from most other gases in being totally absorbed by a solution of sulphate of copper (Gregory). The specific gravity of the gas is 2.695. It contains by weight 96.2 per cent. of arsenic; and as 100 cubic inches would weigh 82.17 grains, every cubic inch will contain more than 8-10ths of a grain of arsenic in a finely divided or gaseous state, and therefore well fitted to penetrate into the blood through the membrane of the air-cells. (*Guy's Hosp. Rep.*, 1860, p. 208.) It is one of the most formidable poisons with which we are acquainted. No treatment can save life when it has been once respired.

CHAPTER XXXVIII.

EFFECTS PRODUCED BY METALLIC MERCURY AS A LIQUID AND IN VAPOR—CORROSIVE SUBLIMATE—TASTE AND SOLUBILITY—SYMPTOMS COMPARED WITH THOSE OF ARSENIC—SLOW OR CHRONIC POISONING—SALIVATION FROM MERCURY AND OTHER CAUSES—CANCRUM ORIS.

METALLIC MERCURY is not commonly regarded as a poison. It has been prescribed and taken in large doses by patients suffering from obstruction of the bowels, without injury to health or causing any uneasiness, except that which might arise from its great weight. In a case of obstruction which proved fatal, half a pound of liquid mercury was swallowed by the patient five days before death. It produced no ill effects, and on an inspection of the body no mercury was found. It had probably been passed by the bowels. In another case of obstinate constipation, a woman, æt. 42, swallowed two pounds of mercury at intervals! The mercury remained nine days in her body, and was perceptible to the feel through the abdomen. The last portions of metal were passed by stool on the fourteenth day; but only five-sixths of the quantity administered were recovered. Slight salivation appeared about this time, but this after-effect was speedily subdued. (Casper's *Wochenschrift*, April 12, 1845, p. 249.) In the same journal, Dr. Kerstein relates a somewhat similar case, in which, under an attack of ileus, he gave to a man, æt. 28, two pounds of quicksilver, in four doses—six ounces at each dose. Croton oil was then prescribed, and after eight days the bowels were moved, the greater part of the metal having been passed unchanged, except some portion which had been converted into black oxide (30 Mai, 1846).

These facts must not lead us to suppose that mercury can, in all cases, be taken with impunity. On some occasions it may undergo chemical changes in the body, and operate as a poison. Sir D. Gibb describes the following case: For the purpose of causing abortion a girl swallowed four and a half ounces by weight of mercury. It had no effect on the uterus, but in a few days she suffered from a trembling and shaking of the body (mercurial tremors) and loss of muscular power. These symptoms continued for two months, but there was no salivation and no blue mark on the gums. (*Lancet*, 1873, vol. 2, p. 329.)

Dr. Brown, of Lahore, states that metallic mercury (*párá*) is often used by the natives of India in order to injure, aggrieve, or annoy those who have given them offence. They think that when mercury gets into the body, it can only come out again through the skin, producing sores and leprous spots. In one case cited by him the question was raised: Is mercury a poison? The sub-assistant-surgeon, to whom the case was referred, stated that in his opinion metallic mercury was not a poison. This stopped the trial. Dr. Brown examined the facts, and very properly dissented from this view. The person to whom the mercury had been given had redness and swelling of the gums, and they bled on pressure with the finger. The woman vomited twice after taking the mercury, and some globules of the metal were found in the vomited matter. A conviction was ultimately obtained by altering the indictment and describing mercury as an "unwholesome drug." (*Medico-Legal Report of Bengal Presidency*, 1869, p. 152.) The technical difficulty which had been raised in reference to the use of the term poison was thus removed. Although liquid mercury is not in itself poisonous, it is liable to be converted into poisonous compounds in the body.

Mercurial Vapors.—Mercury may pass into vapor at all temperatures. In the trades in which this metal is used, it may thus penetrate into the system by slow degrees. The chronic effects are manifested by tremors and paralysis of the limbs—a state called shaking palsy—giddiness, loss of memory, disturbance of the intellectual faculties, salivation and ulceration of the gums, colic, general emaciation, and death A blue line, as in chronic poisoning by lead, may be found at the edges of the gums. Water-gilders, and the manufacturers of looking-glasses, barometers, and thermometers, are subject to these disorders. The frequent contact of mercury with the hands may suffice to produce them in a modified degree. A case, in reference to the noxious effects of mercurial vapor, is reported by M. Chevallier. (*Ann. d'Hyg.*, 1841, vol. 1, p. 389.) It was alleged that two children had suffered seriously in health in consequence of the distillation of mercury being carried on in an apartment below that in which they lived. They had general tremors and others symptoms indicative of mercurial action; but there was no salivation. It has been remarked that those who are subject to mercurial palsy are not very liable to become salivated. M. Chevallier detected mercury in the dust of the apartments on all the floors of the house; and his conclusion was, that the disordered health of the children was certainly due to these mercurial emanations. (See also *Ann. d'Hyg.*, 1847, vol. 2, p. 458.)

A remarkable instance of the noxious effects of mercurial vapor was observed in the case of the Triumph, while conveying a cargo of quicksilver off Cadiz, in April, 1809. By some accident the leathern bags containing the metal burst, and *three tons* of quicksilver were dispersed through the vessel. The crew soon began to suffer from salivation, partial paralysis, and disorders of the bowels. In three weeks no fewer than 200 men were salivated. Two men died from excessive salivation; one lost some of his teeth, and his cheeks were in a gangrenous condition; the other lost the whole of his teeth, the greater part of his tongue, and at the time of his death the lower lip was in a state of gangrene. The interior of the ship was covered with a black powder, and the copper bolts were mercurialized. The vapor proved tal to the animals on board; for nearly all the poultry, sheep, pigs, tice, goats, cats, and dogs, and even a canary bird, died from its inuence. (Paris and Fonblanque, *Med. Jur.*, vol. 2, p. 461.) The oison in this case was not merely the vapor of metallic mercury, but robably in part oxide of mercury produced by friction and diffused as fine dust.

Noxious effects may be apprehended when any operations with netallic mercury are carried on in small and ill-ventilated apartments, eated to a temperature above 70°. The best test for the detection of his vapor is the suspension of a slip of pure gold-leaf in the apartment. If mercury be present, this will become slowly whitened by amalgamation. It is easy to prove by this experiment in a closed vessel that mercury is volatilized at all temperatures.

Blue pill and Mercurial ointment are preparations in which the metal mercury is finely reduced, and probably, as in the mixture of mercury and chalk, more or less oxidized. A case in which a woman is reported to have died from taking excessive doses of blue pill is reported in the *Medical Times and Gazette* (vol. 1, 1863, p. 446). Blue or mercurial ointment, which contains nearly half its weight of mercury, has attracted some attention lately by reason of its poisonous effects on cattle. It is employed for the purpose of dressing sheep in place of arsenic, and so much has been used in Lincolnshire, that Mr. Gamgee informs me that twenty-five tons of this ointment had been sold in one year by a druggist in Boston, chiefly to farmers! In March 1863, I was consulted in reference to the death of many sheep belonging to a farmer near Stamford; it was supposed that the blue ointment employed was not pure, but that it contained corrosive sublimate or some other deadly poison. The sheep were quite healthy, before the ointment was applied as a dressing for the fly; but soon afterwards, they began to die at the rate of six per diem, until upwards of forty were lost. The chief symptoms preceding death were short breathing, with a peculiar grunt indicative of pain, and the heads of the animals drooped to the ground. On inspection, the lungs were generally found congested. On analysis the ointment was found quite pure. Mr. Gamgee informs me that he has been consulted in cases in which sheep have been poisoned by repeated dressings with blue ointment; and he affirms that ruminants are more easily killed by such an application than other animals. He found that the bodies of sheep thus poisoned with mercury had been sent for sale to the dead-meat markets in London, and that they had realized more money than sound mutton sold in the county of Lincoln. I agree with him, that this practice of inunction with mercury should be suppressed: it is not only injurious to cattle, but is often an unsuspected source of noxious food to human beings.

Mercury with chalk (*gray powder*) is commonly regarded as an innocent medicinal mixture of the metal with chalk; but if long kept and exposed to light, a portion of the mercury passes to the highest state of oxidation, and thus produces an irritant or even a poisonous action upon the system. This may account for the severe symptoms which have sometimes resulted from medicinal doses of gray powder. Drs. Duncan and Seeley, who have investigated the changes, state that in one specimen, which should have contained 37.5 parts of metallic mercury, 4.05 parts had become converted into the black oxide, and 22.25 parts into red oxide. I am informed that antimony and arsenic have been detected in this medicine.

Cases of mercurial poisoning are not so frequent as those of poisoning with arsenic.' In England and Wales, in five years (1863–7), fifty-eight deaths were recorded. The most important of the mercurial poisons is CORROSIVE SUBLIMATE.

CORROSIVE SUBLIMATE.

This substance is commonly seen under the form either of very heavy white crystalline masses or of a white powder, and is known by the chemical name of *Chloride*, *Perchloride*, *Bichloride of Mercury* or *Mercuric Chloride.* The term *Chloride* has been and is now by many chemists also assigned to calomel. To prevent any confusion from scientific chemical nomenclature, the old and popular name of Corrosive Sublimate is here retained. This compound is not often taken as a poison. In the coroner's report for 1837–8, there were about fifteen fatal cases of mercurial poisoning, in twelve of which corrosive sublimate was the poison taken. It is chiefly used for the purposes of a bug-poison, also in preserving timber from the dry-rot, and in bronzing gun-barrels. It is freely retailed to the public at the rate of twopence for one or two drachms; if exceeding this quantity, the price is sixpence per ounce. This may guide the witness when he has to judge of the quantity taken, by the price paid.

Taste and Solubility.—The *taste* of corrosive sublimate is powerfully acrid and metallic, so that no poisonous quantity of it could be easily swallowed without the person becoming immediately aware of the fact. It is very *soluble* in water, hot or cold, and speedily sinks in it, in which properties it differs strikingly from arsenic. I have found by experiment that one hundred grains of a cold saturated solution hold dissolved, at a maximum, ten grains of corrosive sublimate; and it is stated by most chemists that two parts of boiling water (212°) will dissolve one part of the poison. The pharmacopœial solution (*Liquor Hydrargyri Perchloridi, B. P.*) used in medicine, contains only half a grain of corrosive sublimate with half a grain of chloride of ammonium to one fluid ounce. Corrosive sublimate is also readily dissolved by alcohol and ether; the latter solvent takes up one-third of its weight, and has the property of abstracting it from its aqueous solution—a principle which is sometimes advantageously resorted to for separating the poison when dissolved in organic liquids. It is soluble without change in nitric and hydrochloric acids.

The solubility of corrosive sublimate in an alcoholic liquid (whisky) was a material question in *Reg.* v. *Walsh* (Kilkenny Summer Assizes, 1850). The prisoner had poured the whisky on the poison in a cup, and the deceased had drunk it. It produced its usual effects. Some testimony was brought to show that the spirit would not dissolve enough to destroy life, but this statement was confuted. The prisoner had artfully given some of the whisky shortly before to another man, who did not perceive any taste, and did not suffer any injurious effects, while the deceased complained that it had a "queer burning taste," and that he felt a burning in his mouth and throat. The whisky was not, in fact, poisoned, but it only became so when poured on the corrosive sublimate in the cup! This gave some plausibility to the defence. (*Med. Gaz.*, 1850, vol. 46, p. 253.)

Symptoms—Acute Poisoning.—The symptoms produced by corrosive sublimate generally come on immediately or within a few minutes after the poison has been swallowed. It differs from arsenic in producing by contact a chemical or corrosive action on the animal membranes. There is perceived a strong metallic taste in the mouth, often described as a coppery taste, and there is, during the act of swallowing, a sense of constriction almost amounting to choking or suffocation, and a burning heat in the throat, extending downward to the stomach. In a few minutes violent pain is felt in the abdomen, especially in the region of the stomach, which is increased by pressure. Pain in the abdomen is, however, sometimes wholly absent. There is nausea, with frequent vomiting of long stringy masses of white mucus, mixed with blood; and this is accompanied by profuse purging, the evacuations being of a mucous character, and in some cases marked or streaked with blood. The countenance is sometimes swollen and flushed, in other cases it has been pale and anxious. The pulse is small, frequent, and irregular, becoming scarcely perceptible when the symptoms are aggravated. The tongue is white and shrivelled, the skin is cold and clammy, the respiration difficult; and death is commonly preceded by syncope, convulsions, or general insensibility. The internal parts of the mouth, with the lips, are white and swollen, and have presented a white or milky appearance as if the cavity had been washed with a solution of nitrate of silver. It is worthy of remark that on a few occasions the tongue and mouth have not presented these changes. Suppression of urine has been frequently noticed among the symptoms. It existed in a well-marked case of poisoning by this substance at Guy's Hospital. The patient lived four days, and did not pass any urine during the whole of this time. (*Guy's Hospital Reports*, April, 1874, p. 24.) This symptom was also observed in a case reported by Dr. Wegeler (Casper's *Wochenschrift*, Jan. 10, 1846, p. 30), in which a youth, æt. 17, swallowed three drachms of the poison, and died on the sixth day. During the last three days no urine was secreted. The case was otherwise remarkable from the fact that no pain was experienced on pressure of the abdomen, and that the pulse underwent no change until shortly before death. In another case, reported by the late Dr. Herapath, in which a scruple of corrosive sublimate in solution was swallowed, suppression of urine and salivation came on, on the third day, and the patient died on the ninth day. (*Lancet*, Dec. 13 and 27, 1845, pp. 650, 698.) In a case observed by Mr. Morris, the quantity of urine secreted was small, and it produced a scalding pain when voided. (*Prov. Med. Journal*, Nov. 18, 1843, p. 126.) In this instance there was no purging.

As contrasted with the effects of arsenic, it may be observed: 1, that corrosive sublimate has a well-marked taste; 2, it produces violent symptoms within a few minutes; and 3, the discharges are more frequently mixed with blood. The symptoms produced by corrosive sublimate, in the first instance, resemble those of cholera; if the person should survive several days, they, in some respects, assume the character of dysentery—tenesmus and mucous discharges mixed with blood, being very frequently observed.

A swelling of the salivary glands and an increased flow of saliva are commonly enumerated among the symptoms, but these are by no means necessary attendants on cases of acute poisoning. Unless the patient survives two or three days, salivation is not commonly observed among the symptoms, and even in these cases it is not always met with. In a case which occurred to Dr. Venables, in which two drachms of the poison had been taken, and the woman survived eight days, there was no salivation. In another, reported by Mr. Wood (*Ed. Med. and Sur. Jour.*, vol. 51, p. 141), in which half a teaspoonful of the poison was taken, salivation was profuse in the course of a few hours. In a case which occurred at Guy's Hospital, in February, 1843, in which two drachms had been swallowed, salivation commenced in four hours; but this is by no means the earliest period. Dr. Percy relates a case, in which the saliva was flowing profusely an hour and a half after the woman had taken a dose of thirty grains. (See *Med. Gaz.*, 1843, vol. 1, p. 942.) In these instances of early salivation, it is alleged that fetor of the breath is absent, and that the salivation is the result, not of absorption, but of a local irritant effect exerted by the corrosive sublimate. But most practitioners will look merely for an effect on the salivary organs. The local action of the poison is, in some instances, sufficient to account for the abundant flow of saliva independently of the influence of absorption. In a case, in which half a drachm of the poison in powder was placed by a woman on her tongue, the saliva soon flowed abundantly from the mouth, and the lips were much swollen. (*Prov. Med. Jour.*, Nov. 18, 1843, p. 127.) This was undoubtedly due to a local effect of the poison. (See also *Guy's Hosp. Rep.*, April, 1844, p. 24.)

As in the case of arsenic, the symptoms caused by this poison are liable to great variation, even when the dose is similar. (See case by Mr. Ward in *Med. Gaz.*, 1848, vol. 41, p. 779.)

In a case which was the subject of a criminal trial, in which an unknown quantity of this poison was given in whisky, the symptoms were of a very marked kind; there was a burning pain in the mouth and throat with immediate sickness; pain in the stomach as well as about the mouth and head; the vomiting was incessant. There was profuse salivation on the third day, with a discharge of shreds of corroded membrane mixed occasionally with blood. The breath was offensive; the tongue swollen and protruding; the teeth were black; the gums and palate ulcerated; the salivation continued, the man became weaker, and before death, on the fifteenth day, there was a discharge of blood from his mouth. (*Med. Gaz.*, 1850, vol. 46, p. 254.)

Chronic or Slow Poisoning.—The symptoms are much modified when the poison is taken in small doses at certain intervals for some days or weeks. There are colicky pains, with nausea, vomiting, general uneasiness, and depression. The salivary glands then become painful, inflamed, and ulcerated; the tongue and gums are red, swollen, and painful, sometimes ulcerated, and there is a peculiarly offensive smell (fetor) of the breath. An examination of the saliva, by a process elsewhere described, will enable a medical jurist to determine whether the salivation depends on mercury or not. The saliva is one medium of elimination, and I have found that mercury may be detected in it after the second or third day, even when the metal has been introduced by inunction through the skin. A blue line, like that observed in poisoning with lead, is sometimes found around the edges of the gums where they join the teeth. The patient experiences difficulty of swallowing and breathing. The constitutional effects are indicated by looseness of the bowels, spitting of blood, cough, general trembling of the limbs and palsy, with low fever and emaciation, under which the patient sinks.

Should the person survive some time, *salivation* or ptyalism is commonly met with. This is one of the most marked effects of slow or chronic poisoning by mercury. In acute cases, it may show itself in persons who survive two or three days, but it is by no means a certain symptom. In chronic cases an increased flow of saliva is almost always present. In placing reliance upon this symptom, it must be remembered that there are persons who are not susceptible of this action of mercury; and further, that salivation may arise from a variety of causes irrespective of mercurial poisoning. In some cases, the salivary glands are easily affected, so that the usual innocent doses of mercurial medicines have been known to produce salivation to such a degree as to cause death. Facts of this kind are of some importance, since charges of malapraxis may be easily raised in respect to them. Dr. G. Johnson met with a case in which three grains of gray powder produced salivation (*Sanitary Record*, July, 1874); and among fatal cases from small doses of mercurial medicines, the following may be mentioned: In one which occurred to Sir R. Christison, two grains of calomel destroyed life by the severe salivation induced, as well as by ulceration of the throat. Another was mentioned to me by a pupil, in 1839, in which five grains of calomel killed an adult by producing fatal salivation. From some cases related by Mr. Samuel, of Newark, it appears that two grains of calomel, divided into three powders, were given in the proportion of one powder daily (two-thirds of a grain), to a little boy aged eight. This small dose produced violent salivation, sloughing, and disease of the jaws, from which he was some weeks in recovering. In another instance a girl, aged five, took daily, for three days, three grains of mercury and chalk power. Her mouth was severely affected, sloughing ensued, and she died in eight days. In a third case, a boy, æt. 11, took three doses of this powder, one of six grains on the 14th, a similar dose on the 17th, and four grains on the 20th, making altogether sixteen grains in a week. Profuse salivation followed, sloughing commenced in both cheeks and rapidly extended through them. The boy died in four days. Previously to taking the mercury the boy had recovered from an attack of fever. (*Lancet*, Dec. 20, 1851, p. 579.) In a fourth case, three grains of blue pill given twice a day for three days, making eighteen grains, were ordered for a girl aged nineteen, who complained of a slight pain in her abdomen. Severe salivation supervened, the teeth separated, and she died in twelve days. With respect to the effects of corrosive sublimate, Sir R. Christison states that he has known three grains only of this substance, in three doses, cause violent salivation. (*Op. cit.*, 408.) When this state results from the use of mild mercurial medicines in small doses, the severe effects may be referred to idiosyncrasy, or a state called intolerance (*ante*, p. 71). A person may die under these circumstances, either from simple exhaustion, or from excessive sloughing of the throat with disease of the bones. When a patient has recovered from the first effects of acute poisoning by corrosive sublimate, he may die at almost any period from these secondary consequences.

Some quack medicines contain mercurial compounds in such quantity as to occasion fatal salivation. Thus, what are popularly termed *Worm Lozenges*, have destroyed life under these circumstances. In December, 1853, a lady, æt. 46, took one of these lozenges for four nights in succession. In three days she suffered from salivation, and her tongue and gums were much swollen. In spite of treatment, she gradually sank and died from the effects.

In *Jones* v. *Fay* (Croydon Autumn Assizes, 1865), an action was brought by plaintiff against a druggist for supplying him with pills containing mercury (blue pill), whereby his health had been injured, owing to profuse salivation. It appeared from the evidence that plaintiff had suffered severely from lead-colic. He received from the defendant some pills, which he continued to take for some time. He was seen by a medical man, who found him suffering from profuse salivation, as the result of mercury. His tongue was swollen and protruded; the salivary glands were red and painful, and he complained of pain in his limbs as if he had been beaten. A short time afterwards some of the pills were analyzed, and found to contain mercury in the form of blue pill. Drs. Guy and Harley deposed that mercury was an improper medicine in a case of lead-colic, and that the plaintiff's health had been destroyed by the effects of the mercurial medicine. In defence, it was urged that no mercury had been given, but the jury declined to adopt this theory, and returned a verdict for the plaintiff, with £100 damages. There was no cause for the salivation but the mercury, and this had so exhausted the plaintiff that he died within twenty-four hours after a verdict had been returned in his favor. The evidence of salivation was clear enough in this case, but as the composition of the pills was disputed, and they were not analyzed until some time after the action had been commenced, it would have been more satisfactory if the saliva had also been submitted to analysis.

It is generally admitted by toxicologists, that salivation may be intermittent, *i. e.*, that it may cease and reappear without more mercurial poison, or any mercurial preparation, being given in the interim, although such cases are rare. As a matter of medical jurisprudence, this question was brought to an issue at the trial of Butterfield, at Croydon, in 1775. The deceased was supposed to have been killed by the administration of small doses of corrosive sublimate, and the fact of his having been salivated at or about the time of the alleged administration, was regarded as a proof of poisoning. In the defence, it was urged that the deceased had been salivated two months previously, under a common mercurial course, and although the salivation had ceased for that period, it was probable that this was nothing more than a recurrence of the former—it did not prove that there had been any fresh administration of mercury in the interim. There was a difference of opinion on this point among the witnesses, as there probably would be in the present day, if each relied upon his own personal experience. However, one of the witnesses stated that he had known salivation to recur without a fresh exhibition of mercury after the long interval of *three months*, and the prisoner was acquitted. Cases are reported of salivation recurring after intervals even longer than this. One is quoted by Mr. Swan, in which salivation recurred after an interval of six months. (*On the Action of Mercury*, 1847, p. 4.)

It is proper to bear in mind that salivation is not necessarily connected with the administration of mercury, and therefore, when taken alone, it can never furnish proof of mercurial poisoning. Salivation may come on *spontaneously* as a result of disease in the salivary organs; or it may arise from simple mechanical causes. Dr. Mulock communicated a case to the *Dublin Hospital Gazette*, in which profuse salivation was occasioned by the introduction of a set of artificial teeth. (Sept. 15, 1845, p. 35.)

Salivation has sometimes appeared in a severe form in the early stage of pregnancy. Dr. A. Farre met with a case of this kind in a woman who had had four children, and was pregnant with a fifth. Without any apparent cause profuse salivation showed itself in the second month. The saliva streamed from her mouth in a quantity amounting to three pints a day. No mercury had been given, the salivary glands were not enlarged, and there was no fetor of the breath. This symptom passed off after quickening, and the woman's health was restored. (*Trans. of Obstet. Soc.*, vol. 15, 1874, p. 222.)

Salivation has been known to be produced by many medicinal substances besides mercury. Thus it has been known to follow the use of the preparations of gold, copper, bismuth, lead, antimony, iodine, iodide of potassium, croton oil, opium, prussic acid, carbolic acid (*Guy's Hosp. Rep.*, 1870, p. 533), sulphuric acid, arsenic, colchicum, foxglove, and cantharides. Some have asserted that an offensive odor of the breath, a brassy taste in the mouth, and spongy and ulcerated gums, will indicate the salivation caused by mercury; but these characters have been equally met with in the salivation produced by arsenic and bismuth. (*Prov. Med. Journ.*, Oct. 22, 1845, p. 638.) A case in which this question was material has been reported by Mr. Harding. (See *Lancet*, June 13, 1846, p. 654.) The true criterion of mercurial salivation is the detection of mercury in the saliva. From recent observations it appears that the metal is eliminated by all the fluid secretions, but chiefly by the urine, saliva, and the mucous fluids of the intestines. (*Lancet*, 1873, vol. 1, p. 476.) In doubtful cases of chronic poisoning the examination of the uripe may prove a great aid to diagnosis.

It appears from the researches of pathologists, that salivation is not so readily induced by mercurial preparations in young persons as in adults. (See *Dubl. Med. Press*, May 12, 1847, p. 296; also, *Amer. Journ. Med. Sciences*, April, 1874, p. 509.)

In addition to the facts already detailed, respecting death from excessive salivation under the use of small doses of mercury, there are certain *morbid* states of the body which appear to have the effect of increasing this action of the medicine on the salivary glands. This kind of acquired idiosyncrasy exists especially in that form of disease called granular degeneration of the kidney, which is characterized in its early stage by albuminuria, Dr. Craigie has observed, that when given to persons laboring under symptoms of granular kidney, a small quantity of mercury induces salivation, and renders the mouth tender and most painful. (*Practice of Physic*, ii, 1148.) Sir R. Christison has repeatedly observed that mercurial action (salivation) is in these cases brought on by unusually small doses of the compounds of mercury, or unusually soon. A medico-legal case involving this question occurred at Reading in December, 1845. A man, laboring under disease of the kidneys, had placed himself in the hands of a person who promised to cure him. Part of the treatment consisted in the administration of small doses of mercury. Profuse salivation came on, and the patient, not finding himself relieved, applied to a medical practitioner. In about a fortnight afterwards the man died, and a coroner's inquest was held, in order to determine whether he had not died from improper treatment. It appeared in evidence that some calomel pills were prescribed, and that had the prescription been followed, the deceased would have taken no more than six grains in the five days that he was under treatment; but, in consequence of some mistake, he took *eleven grains and a quarter*—*i. e.*, two grains and a quarter daily for five days. On an inspection of the body, the gums were found ulcerated, and the mucous membrane of the tongue, mouth, and throat, was in a state of intense irritation. Both kidneys were enlarged and in a diseased state. After hearing the evidence of several medical witnesses, the jury returned a verdict that the deceased had died from natural causes.

Cancrum Oris—Canker of the Mouth.—Corrosive sublimate, as well as other mercurial preparations, is liable to produce gangrene of the mouth and throat, and thus destroy life. A disease called *canker*, or gangrene of the mouth, attended with ulceration of the gums and a falling out of the teeth, has been observed to occur in infants and children, to whom no calomel, nor any mercurial preparation whatever, had been given. Those who especially suffer from this disease are children badly fed and clothed, and generally laboring under, or recovering from fever, small pox, measles, or hooping-cough. It is, however, far more common as a consequence of measles than of other diseases, and it is always connected with a depressed state of the vital powers. Several cases of cancrum oris have been reported by Dr. Hennis Green. (See *Lancet*, Dec. 1839.) On these occasions, supposing any mercurial preparation to have been given medicinally, it may become a serious question whether death actually resulted from the mercury acting as a poison, or from the effects of disease. In some fatal cases that have occurred, the subject has become a matter of inquiry before coroners. Although salivation and its consequences—ulceration of the gums and sloughing—are not common among children as effects of mercury, yet it is clear, from the facts already cited (p. 356), that small doses of mercurial medicines may have occasionally a most violent effect upon them, and render the suspicion of mercurial poisoning probable. Of two children, whose deaths were the subject of investigation under these circumstances, one was affected with hooping-cough, and the other with measles. Powders containing calomel were prescribed in both cases—gangrene of the mouth supervened, and the children died. There was some reason to believe, from the evidence, that the mercurial medicine had really produced the effect attributed to it, at least in one of the cases. In August 1840, a charge was made against a medical practitioner of having caused the death of a child, aged four years, by administering an overdose of some mercurial preparation. The child was laboring under hooping-cough, and some medicine was prescribed. On the fourth day the child complained of soreness of the mouth, the teeth became loose and fell out, the tongue and cheek were much swollen, and the child died in the course of a few days from gangrene in the left cheek. The answer to the charge was, that not a particle of mercury had been exhibited—a fact clearly proved by the production of the prescription-book of the medical attendant. This, then, was an instance in which gangrene from spontaneous causes (*cancrum oris*), was mistaken for mercurial poisoning. Had the medicine prescribed contained any mercury, a verdict affecting the character of the practitioner would probably have been returned! (See also a case by Mr. Dunn, *Med. Gaz.*, vol. 33, p. 57; and *Br. and For. Med. Rev.*, October, 1844, p. 542.) Dr. Dugas considers that children between five and eight years of age are especially liable to this form of mercurial salivation. (*Ed. Monthly Journ.*, May, 1851, p. 481.) It is worthy of remark, that in fatal cases, the popular opinion is generally supported by that of some medical practitioner; showing how easily members of the profession, as well as the public, are led to refer the effects to what, in many instances, is only an *apparent* cause. An important case of this kind, in which the medical witness relied upon the "mercurial fetor" as characteristic and distinctive, will be found in the *Lancet* (June 13, 1846, p. 654.) One of the most common forms of chronic poisoning by mercury is seen in the action of mercurial vapor. (See p. 352, *ante.*)

CHAPTER XXXIX.

CORROSIVE SUBLIMATE—FATAL EFFECTS OF EXTERNAL APPLICATION—ABSORPTION OF CORROSIVE SUBLIMATE BY THE SKIN—APPEARANCES IN THE BODY AFTER DEATH—QUANTITY REQUIRED TO DESTROY LIFE—FATAL DOSE—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT OF POISONING WITH CORROSIVE SUBLIMATE.

CORROSIVE SUBLIMATE.

Effects of External Application.—Cases of poisoning by the *external* application of corrosive sublimate are not very common. It acts through the unbroken skin, and more powerfully through ulcerated surfaces, producing severe local and constitutional symptoms and even death. Two fatal cases of this kind occurred to Mr. Ward, of Bodmin. (*Med. Gaz.*, vol. 3, p. 666.) A man, aged 24, rubbed over every part of his body one ounce of corrosive sublimate, mixed with six ounces of hog's lard, for the purpose of curing the itch. In an hour he experienced excruciating pain in the abdomen and over the whole of his body; he said he felt roasted alive; he also suffered from intolerable thirst. The skin was found completely vesicated. He died on the eleventh day, having labored under bloody vomiting, purging, and tenesmus (straining). Salivation did not show itself until thirty-six hours after the application of the poison. The brother of the deceased, aged 19, rubbed in the same quantity of the poison. The symptoms were much the same, but more aggravated. There was constant vomiting, with complete suppression of urine, and frequent bloody evacuations;—the salivation was not so severe. He died on the fifth day. On inspection the stomach was found much inflamed and partially ulcerated. The small intestines were also greatly inflamed throughout, and the lower portion of the colon and rectum were in a state of mortification. The bladder was contracted, and without urine. Thirty large worms were found alive in the stomach and intestines! (For another case see Niemann, *Taschenb. der Arzneiw.*, p. 452.) Mr. de' Ricci, of Ballymahon, met with two cases somewhat similar to those of Mr. Ward. Two boys, æt. 11 and 7 respectively, were treated by a quack for scalled head. This man, it appears, rubbed on the diseased surfaces an ointment consisting of two drachms of corrosive sublimate and one ounce of tallow. It produced immediately the most intense suffering, and in from thirty to forty minutes there was vomiting, with pain in the bowels, followed by purging and bloody evacuations. The boys continued to get worse until death. The younger died on the seventh day, and the elder on the ninth. There was no salivation in either case; in the younger child there was an appearance like *cancrum oris* (*ante*, p. 360); in the elder there was a rash, like the eczema of arsenic, about the mouth. In the younger child there was a complete suppression of urine, while in the elder it was merely diminished in quantity. On inspection the morbid changes were chiefly confined to the stomach and bowels. The mucous membrane of the stomach was injected with red blood throughout: but there was no ulceration or softening. In one there were a few spots of effused black blood in addition to this injected appearance. In both the intestines were highly inflamed and ulcerated: and in the younger there were twenty-three intussusceptions (*Chemist*, 1854, p. 760; and *Dublin Quarterly Journal*. Aug. 1854).

Death from the external application of corrosive sublimate has been the subject of two criminal trials. In both there were the usual symptoms of irritation, and the stomach and intestines were much inflamed. (*Reg.* v. *Welch*, Worcester Summer Assizes, 1845; *Med. Gaz.*, vol. 36, p. 608, and *Reg.* v. *Crook*, Winchester Lent Assizes, 1859.) In the last case a quack was convicted of manslaughter by applying corrosive sublimate in powder in improper quantity to a cancerous tumor on the face of the deceased. The poison was absorbed, and produced the usual symptoms and appearance. Mr. May, of Reading, detected corrosive sublimate in the diseased part.

The readiness with which this poison acts through the sound skin is proved by the following circumstance. M. Cloquet plunged his hands into a concentrated solution of corrosive sublimate, in order to remove some anatomical preparations. He did not wash his hands afterwards; and in about eight hours he was attacked with severe pain in the abdomen, constriction in the chest, painful respiration, thirst, nausea, and ineffectual attempts at vomiting. Under the use of diluents these symptoms were removed, but for eight days he suffered from pain in the stomach. (*Galtier*, vol. 1, p. 567.) There is reason to believe that, in respect to themselves and their patients, medical men are not sufficiently aware of the absorbent powers of the unbroken skin in contact with this poison. One of my pupils, now in practice, informed me of two cases in which medical men applied lotions of corrosive sublimate to their skins. In one instance the corrosive sublimate was used in the proportion of eight grains to one ounce of spirit. It produced immediate and intense irritation, followed by vesication and suppuration. He suffered for several days from irritative fever and tenesmus. In the second case from one to two fluid drachms only of a similar solution were used, and the parts were immediately bathed. In spite of this, similar local and constitutional symptoms followed. Physicians of repute occasionally employ a solution of corrosive sublimate in the treatment of skin diseases, but the dangerous results of this practice are well indicated by the following case, which occurred in Sept. 1871.

A girl, æt. nine, was suffering from ringworm spreading over the scalp. The physician in attendance applied to the scalp a liquid, consisting of eighty grains of corrosive sublimate, dissolved in an ounce of alcohol. In a few hours, the usual symptoms of acute mercurial poisoning set in. There was great pain, with œdematous swelling of the face and head, restlessness, sickness, and purging. The gums were swollen and tender, as well as the mouth and lips. Vesications even formed on the head, and there was salivation. The girl died on the fifth day, obviously from mercurial poisoning, as a result of absorption through the skin. At the inquest, the medical gentleman who had made use of this highly poisonous solution to the scalp is reported to have said that its effect was always local, and that corrosive sublimate thus applied to the skin was never absorbed into the system! The symptoms and death were referred by him to idiosyncrasy. The jury at the inquest were not satisfied with this explanation, they returned a verdict that deceased had died from poison, and they censured the medical man for his treatment. (*Pharm. Journal*, Sept. 9, 1871, p. 216; *Lancet*, 1871, vol. 2, p. 473, and *Med. Times and Gaz.*, 1871, vol. 2, p. 353.)

The strange part of this case is that any educated medical man should doubt that poisoning by corrisive sublimate can take place by absorption through the skin, whether broken or unbroken, or that he should be prepared to assign the fatal effects to idiosyncrasy. A solution in alcohol containing three fatal doses in a teaspoonful can hardly be regarded as a safe application. The cases above related show that the jury took a common-sense view of the matter. Such a case as this should onçe for all operate as a caution to medical men in the local use of this powerful agent.

Mr. Annan has reported an instance in which the local action of corrosive sublimate appears to have led to death after a long period. In Jan. 1845, a shepherd, æt. 38, had been employed several hours daily in washing sheep affected with cutaneous disease, with a solution of two drachms of corrosive sublimate in twenty ounces of water, in which muriate of ammonia was also dissolved. He was suddenly seized with sickness, vomiting, constitutional irritation, and after the lapse of five days, with salivation, although not severe. He did not recover for a fortnight. In six weeks he experienced a similar attack from the same cause, and this left behind it great debility and emaciation. He resumed his occupation, but was attacked with wandering pains in the joints and diseases of the bones, as if from the secondary effects of mercury; and he died fourteen months after the first attack. (*Med. Times*, July 25, 1846, p. 331.) Of ten of the sheep, two died shortly after the application.

Salivation is a common effect of the external application of this poison. Dr. Guerard has seen ptyalism produced as a result of three corrosive sublimate baths (one ounce of the poison to about ten gallons of water), taken at intervals of three days; but the effects produced by the solution are never so powerful or so dangerous as those which arise from the application of the poison in the form of ointment. There are many ointments sold by quacks, for the treatment of skin diseases, which contain corrosive sublimate.

When any mercurial preparations are used as caustics, salivation may speedily follow. Breschet observed this effect in twenty-four hours from the application of the acid nitrate of mercury to the cervix uteri.

Appearances after Death.—These, as in the case of arsenic, are chiefly confined to the stomach and bowels. Corrosive sublimate, however, affects the mouth, throat, and gullet. The mucous membrane is softened, of a white or bluish-gray color, and sometimes inflamed; in advanced cases, it is found peeling off; that which lines the œsophagus is similarly affected, and partially corroded and softened. The mucous membrane of the stomach is more or less inflamed, sometimes in patches; and there are masses of black extravasated blood found beneath it. The whole cavity is stated to have sometimes presented a slate-gray color from the partial decomposition of the poison by the membrane itself; and beneath this the mucous coat may be found reddened. This gray tint of the mucous membrane has been considered by some to be a special indication of the action of the poison on the living mucous membrane; but it is not always present. The slate-gray tint described by Orfila, and delineated by Roupell, from their experiments on animals, has been only occasionally observed; and it is a matter for consideration whether it may not be one of the appearances which are liable to mislead those who rely strongly on the results of experiments on animals. (See *Ann. d'Hyg.*, Juillet, 1858, p. 204.)

A case occurred at Guy's Hospital, in which the mucous membrane was simply inflamed, and resembled the condition presented in cases of arsenical poisoning. In a case which proved fatal on the fifteenth day, the mucous membrane had a dull slaty appearance. In another case the inner coat presented a deep yellow tint (from bile), with only a slight redness of the folds. M. Lassaigne describes a fatal case in which the stomach had a deep violet-red color, and there was an effusion of blood in the course of the vessels; but there was no ulceration. The coats of the stomach are sometimes corroded, and so much softened that they cannot be removed from the body without laceration. Similar appearances have been met with in the intestines, especially in the cæcum. In Dr. Herapath's case, in which a scruple was taken, and death occurred on the ninth day, the mucous membrane of the stomach was softened, but there were no well-marked appearances of the chemical action of the poison in this organ. The cæcum had been the seat of the most violent inflammation, the whole surface being of a dark-red color, and there were patches of sloughing in the coats. (*Lancet*, Dec. 27, 1845, p. 700.) In the case of a man, æt. 42, who swallowed, by mistake, thirty grains of the poison dissolved, and who died on the twelfth day, the stomach was found empty, and the mucous membrane was of a dull, dark-red color, chiefly about the smaller curvature. It was softened, and near the intestinal end it was gray, pulpy, and gangrenous. In the gullet, the lining membrane appeared to have been stripped off in shreds. The intestines were in a state of intense inflammation, passing into gangrene. The other viscera presented no particular appearance. In this case the symptoms were manifested in a few minutes; there was a burning pain down the gullet to the stomach, described as if the parts were on fire; there was no mark of corrosion in the mouth; there was a sensation as if the throat were "grown up;" and there was blood in the vomited matters as well as in the evacuations. There was no salivation at any period. (*Med. Times and Gaz.*, Feb. 26, 1859, p. 210.) A case occurred to Dr. Thompson, of Perth, in which a man died forty hours after having swallowed two drachms of corrosive sublimate in powder. The mucous membrane of the stomach, duodenum, upper portion of the ileum, and parts of the large intestines, were found of a bright-red color. This appearance was most marked at the cæcum and sigmoid flexure of the colon. The local action of the poison on the mouth and fauces was in this instance considerable. There was no suppression of urine. (*Edinburgh Monthly Journal*, Dec. 1851, p. 532.) Perforation of the stomach is rare as an effect of this poison. There is, I believe, only one case recorded in which this appearance was found. Certain morbid changes have been met with in the urinary and circulating organs; and Mr. Swan states that he has found the ganglia and branches of the sympathetic nerve inflamed; but these changes cannot be regarded as characteristic of this variety of poisoning. Appearances in the alimentary canal, like those just described, have been seen not only where the case has terminated fatally in a few hours, but where it has been protracted for six, eight, and even eleven days. (Chaussier, *Recueil de Mémoires*, p. 363.) In chronic cases inflammation of the salivary organs, with ulceration of the gums, is met with.

In a case which proved fatal on the fourth day, the body was inspected sixteen hours after death. The lungs were somewhat consolidated, as if from early pneumonia. The heart was healthy, and its cavities were filled with colorless firm fibrin. The kidneys were congested. The bladder was empty and contracted, and there were some small pink spots on its mucous membrane. The mucous membrane of the gullet had a vermilion hue. The stomach presented a pink color on its inferior surface, near its middle. The small intestines were healthy, and lined with a thick yellow mucus. The cæcum and ileocæcal valve showed signs of the most intense inflammation; some portions were of a deep purplish-black color, with patches of sloughing mucous membrane, tinged green by fæces. The colon and rectum also exhibited traces of the most violent inflammation, especially the ascending and transverse portions. Here were found oval patches of sloughing mucous membrane, about the size of small almonds, and tinged green by fæces passing over them. The condition of the cæcum here described has been found in other cases.

In another case the lining membrane of the mouth and gullet was quite healthy. The mucous membrane of the stomach, to the extent of three inches from the cardiac opening, was converted into a gangrenous mass, having a corroded, ragged appearance, of a dusky brown color, approaching to black. Around this the mucous coat was reddened, but it was healthy towards the intestinal opening. There were no morbid changes of any note in the intestines. The cavities of the heart were empty. The whole of the mucous membrane of the air-passages was in a state of extreme congestion, varying from a deep red to a purple color; the smaller air-tubes being filled with a frothy, bloody fluid. As there had been no sign of cerebral disturbance in this case, the head was not examined. (*Med. Gaz.*, vol. 41, 1848, p. 780.)

Fatal Dose.—It is difficult to state this with any degree of certainty, since it is only by accident that the quantity taken can be ascertained, and the fatal effects must vary according to many circumstances. A child, aged 3 years, died in twenty-three days from the effects of twelve grains of corrosive sublimate. The *smallest* dose which is reported to have destroyed life was *three* grains. This was also in the case of a child, and the quantity was accurately determined from the fact of its having been made up by mistake for three grains of calomel, which the physician intended to order. (This case is referred to in the *Lancet*, 1845, p. 297.) A very loose and imperfect report, either of the same or of a similar case, is given in the *Ann. d'Hyg.*, 1835, vol. 1, p. 225. It is there stated that three children lost their lives. In the case of *Reg.* v. *Robertshaw* (Carlisle Lent Assizes, 1845), there is reason to believe that two, or not more than three grains, were taken, and proved fatal to an adult. (*Med. Gaz.*, vol. 35, p. 778.) In its power as a poison, it is therefore somewhat similar to arsenic. Persons have been known to recover who have taken very large doses, when remedies were timely administered, or when there was early vomiting. (*Med. Times and Gaz.*, Feb. 18, 1860, p. 162.) I have elsewhere reported a case of recovery from a dose of nineteen grains in a girl, æt. 18. (*Guy's Hosp. Rep.*, 1850, p. 213.) In an instance reported in the *Journal de Pharmacie*, a man recovered in three days after having taken one drachm of the poison; and a case of recovery from a similar dose is described in the *Edinburgh Monthly Journal*, 1850 (p. 380). In the *Medical Gazette* (vol. 14, p. 63), Dr. Booth mentions a case in which *an ounce* of corrosive sublimate had been swallowed after a full meal; and by timely vomiting the subject of this rash act escaped with comparative impunity. In a case by Dr. Percy (*Med. Gaz.*, vol. 31, p. 942), a girl, aged 17, mixed thirty grains in coarse powder with water in a teacup, and then swallowed the liquid. A considerable quantity remained in the cup. Symptoms of poisoning came on, but the girl recovered. Dr. Percy doubted whether any of the poison had reached the stomach. A case of recovery after *forty grains* had been taken in whisky, under circumstances favorable to its fatal operation—*i. e.*, on an empty stomach—is recorded by Dr. Andrews. (*Cormack's Journal*, Feb. 1845, p. 102.) The patient was a woman, æt. 65.

The actually smallest dose required to destroy an adult, under ordinary circumstances, cannot therefore be determined at present from any reported facts. The medicinal solution is used in doses varying from one thirty-second part to one-eighth of a grain. Hence, in potency, corrosive sublimate may be considered as not inferior to arsenic; and a fatal dose, under circumstances favorable to its operation, may be assumed to be from *three* to *five grains*. Three grains have proved fatal to a child.

A case occurred in Guy's Hospital in November, 1861, in which *five* grains, taken dissolved, destroyed the life of an adult in six days. A case is quoted by the late Dr. Beck, in which six or eight grains destroyed the life of an adult. (*Med. Jur.*, vol. 2, p. 570.)

Period at which Death takes place.—In an acute case of poisoning with this substance a person commonly dies in from one to five days. But death may take place much sooner or later than this. A man has died from the effects in three hours and a quarter.

The case occurred to Dr. Skegg in September, 1861 (*Lancet*, February 1, 1862). A man, æt. 54, swallowed one hundred and twelve grains of corrosive sublimate at 11 A.M. When seen by Dr. Skegg, soon afterwards, he was on the bed in a state of great prostration; his skin was blanched and covered with a cold, clammy perspiration; he vomited a thick stringy albuminous-looking substance. There was intense pain over the abdomen, and great purging with discharge of blood; the pulse was scarcely perceptible, the mucous membrane of the tongue and of the interior of the mouth was perfectly white from the local action of the poison. White of egg was given freely, and a mustard poultice applied to the abdomen. At one o'clock he was more depressed. At a quarter past two, Dr. Skegg again saw him, and found that he had just expired. An inspection was made twenty-four hours after death. The external coat of the stomach was of a deep red color. The mucous membrane internally had the appearance of a piece of dark crimson velvet, owing to the large quantity of blood extravasated. The intestines here and there were reddened, The great omentum for about an inch from the stomach was of a deep crimson hue. The other organs were healthy.

In the second case, in which only *five grains* of the poison, dissolved in vinegar, were swallowed by a man, æt. 25, the following symptoms were observed on his admission into Guy's Hospital. Immediately after swallowing it, he felt a burning heat in his throat, and vomited freely. In two hours there was great pain in the abdomen; he passed blood in his evacuations, and brought up a thick yellow frothy matter, tinged with blood. There was suppression of urine. He died on the seventh day. On inspection, the gullet presented marks of the local action of the poison. The mucous membrane of the stomach was reddened, and throughout minutely injected. There was no appearance of chemical corrosion. The small intestines at their lower part, as well as the large intestines, were deeply injected. The cæcum was but slightly affected. Seven ounces of the liver, and one-half of the stomach, gave only slight traces of mercury. The greater part of the poison had no doubt been thrown off by vomiting. It will be observed that in spite of the removal of the poison from the stomach, and its almost entire disappearance from the body, the case proved fatal. (*Guy's Hosp. Rep.*, 1864, p. 183.) Dr. Eade mentions a case in which a man swallowed a lump of corrosive sublimate; it was ejected from his stomach in about an hour, and it then weighed about one drachm. The usual symptoms of mercurial poisoning followed, with suppression of urine. There was slight salivation on the fifth day, and the man died on the eighth day. Mercury was found in the liver. (*Lancet*, 1870, vol. 1, p. 303.) A child, aged 7, was killed in three hours by eighteen grains of corrosive sublimate. In the following instance, reported by Mr. Illingworth, the period, although inferential, was probably even shorter. A man, æt. 30, was found dead on December 4, 1842, at half-past seven A.M. He had vomited some half-digested food, mixed with blood and mucus. On a shelf near him was a drinking-horn, containing about three drachms of corrosive sublimate. It was ascertained at the inquest that he had died from the effects of this poison. He had put water into the drinking-vessel, and had probably swallowed the poison while thus loosely suspended; the exact quantity taken could not therefore be ascertained. The deceased was last seen alive at half-past eleven the previous evening—*i. e.*, only eight hours before he was found dead. When discovered, the face and limbs were cold. From all the circumstances it was inferred that, even admitting the deceased to have taken the poison immediately after he was last seen alive, he could not have been dead for less than six hours. This would carry the duration of the case to *two hours* from the time of taking the poison. (*Med. Gaz.*, vol. 31, p. 557.)

The most rapidly fatal instance of poisoning by corrosive sublimate hitherto recorded, was communicated to me by Mr. Welch. In June, 1846, a man mixed some corrosive sublimate (quantity unknown) with tea, and drank it. The symptoms which followed were a sensation of burning heat in the mouth, and mucous vomiting. He was insensible when seen; and from the circumstances of the case, he must have died in less than *half an hour*.

On the other hand, a case may be protracted for several days. The following summary will not only show this, but will also prove that the time at which the poison destroys life cannot be inferred from the quantity taken. In an instance referred to by Niemann (*Tasch. d. Arzneiw.*, p. 452), one ounce of the poison was swallowed, and the person did not die until the sixth day. In a case related by Dr. Venables, two drachms of the poison killed a woman in eight days. In one reported by Sobernheim, three drachms did not kill for eleven days. A case is quoted by Beck (*Med. Jur.*, vol. 2, p. 570), in which a man who had taken only six or eight grains in solution, survived until the twelfth day. In the *Edinburgh Monthly Journal* (1860, vol. 1, p. 958), a case of similar duration is reported. The case was somewhat peculiar. The dose taken was from sixty to eighty grains of corrosive sublimate. On the first day there was no complaint of pain in the throat or stomach; there was soreness and pain in the throat on the second day, and the mouth and gums were affected on the third day. On the eighth day the man had apparently recovered, but he gradually became weaker, and died on the twelfth day. The most protracted case of acute poisoning which I have met with is that which is elsewhere referred to (*ante*, p. 365), in which death did not take place until the *fifteenth day*. In death from chronic poisoning, the case may be protracted almost indefinitely.

Treatment.—If vomiting does not already exist, it must be excited by the use of emetics. (See TREATMENT OF ARSENIC, *ante*, p. 303.) Various chemical antidotes have been suggested for this poison; and among these, albumen, both of the yolk and white of egg, mixed with water and administered in large quantity, is perhaps the best fitted to counteract its effects. This remedy appears to have been beneficial even when it was not taken until some time after the poison had been swallowed; but too much reliance must not be placed on it. The removal of the poison from the body should be the great object of our treatment. Gluten has also been used with benefit. This may be prepared by washing flour in a muslin bag under a current of water. Should the case be urgent, the flour may be at once exhibited in the form of a thick paste mixed with milk or water. Gluten may often be obtained when albumen is not at hand. M. Bouchardat states that Cullerier saved two hundred patients who had taken an overdose of corrosive sublimate, by making them swallow, in twenty-four hours, from seven to eight quarts of milk, with a decoction of linseed and warm water! (*Gaz. Med.*, Jan. 9, 1847.) These antidotal liquids may be serviceably employed for the purpose of favoring the expulsion of the poison by vomiting, on which the safety of the patient essentially depends.

Sometimes recovery has been wrongly attributed to the remedy employed. In May, 1862, a man swallowed eighty grains of corrosive sublimate dissolved in whisky and water. In ten minutes violent vomiting occurred. A mixture of albumen and milk was first given, and in about twenty-five minutes, gold-leaf with reduced iron made into a bolus. Some warm water had been previously administered in order to clear the stomach of any albumen or mucus. Vomiting recurred with less violence, the matters being mixed with gold-leaf. There was no salivation, and in about eight days the man perfectly recovered. (*Am. Journ. Med. Sci.*, April, 1863, p. 340.) Dr. Johnston attributed the recovery to the gold and iron, but there is not the slightest evidence that the metals had exerted any galvanic action in decomposing the corrosive sublimate; on the contrary, the particles of gold-leaf rejected after the administration of the antidote were apparently unchanged. The recovery was, no doubt, due to the early vomiting, and the free use of albumen and milk.

In all cases, the entire expulsion of the poison from the stomach should be looked to by the practitioner; and albumen or gluten may be given at the same time to aid the efforts of vomiting. The use of the stomach-pump is of questionable propriety; since if the mouth, gullet, and stomach are much softened and corroded, very slight force in its employment might lead to perforation. In order to check excessive salivation, as a subsequent symptom, Mr. Allison has recommended small doses of chlorate of potash. (*Med. Gaz.*, vol. 31, p. 953.)

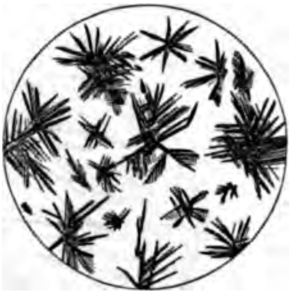
CHAPTER XL.

CORROSIVE SUBLIMATE—CHEMICAL ANALYSIS IN THE SOLID STATE—IN SOLUTION—REDUCTION—LIQUID TESTS—SEPARATION FROM ORGANIC LIQUIDS—DIALYSIS—DETECTION OF MERCURY BY COPPER—BY GOLD AND ZINC—ABSORBED AND ELIMINATED MERCURY—DETECTION IN THE URINE—WHEN MIXED WITH ARSENIC—ABSENCE OF MERCURY FROM THE TISSUES—QUANTITATIVE ANALYSIS.

CORROSIVE SUBLIMATE—ANALYSIS.

In the Solid State.—Corrosive sublimate is usually seen as a *solid* in heavy translucent crystalline masses, or in the form of a white crystalline powder. 1. When the powder is heated on platinum-foil or mica, it melts, and is volatilized in a white vapor without leaving any residue. 2. When heated in a close tube, it melts and forms a sublimate, consisting of prismatic crystals in stellated groups. (Fig. 27.) 3. The powder is changed in color by the following reagents: iodide of potassium produces a bright scarlet, potash a yellow, and sulphide of ammonium a black precipitate; ammonia does not alter its color. 4. The mercury and chlorine may be discovered by one process. Mix the powder with six parts of dried carbonate of soda (obtained by incinerating the bicarbonate), until the residue in the reduction-tube fuses and becomes quite white. A sublimate of metallic mercury in distinct and well-defined globules will be obtained. (Figs. 30 and 31, p. 372.) The *weight* of the sublimate may be determined by the same method as that of arsenic (p. 307, *ante*), and it may be preserved in like manner, *i. e.*, by hermetically sealing the tube. Detach, by a file, the end of the tube containing the fused residue, which is chloride of sodium with some undecomposed carbonate. Digest it a little in water with nitric acid, and apply heat until the residue is entirely dissolved; then add to the solution nitrate of silver. A white precipitate of *chloride* of silver, insoluble in nitric acid, will be at once produced. The solid is thus proved to contain both mercury and chlorine, and the only compound of these elements soluble in water is corrosive sublimate.

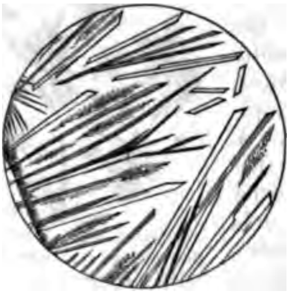
FIG. 27.



Stellated crystals obtained by heating corrosive sublimate, magnified 30 diameters.

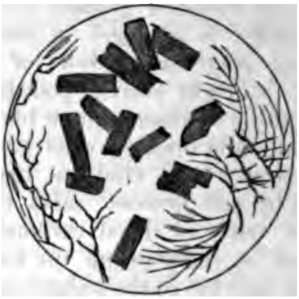
In Solution.—Corrosive sublimate is dissolved by water, alcohol, and ether. One part of the poison is dissolved by sixteen parts of water, three of alcohol, and four of ether. A few drops of the aqueous solution evaporated on a glass-slide yield slender opaque silky prisms (Fig. 28). The solution has a faintly acid reaction on test-paper, and a strong metallic taste. The crystals obtained from the alcoholic solution are larger and better defined (Fig. 29). When a weak solution of iodide of potassium is dropped on them, they acquire a bright scarlet color, and chloride of potassium is formed. These characters, which may be obtained from the minutest crystal and only one drop of solution, prove that the body dissolved in water is corrosive sublimate; it is thus distinguished from every other mineral poison, and all other substances whatever. 1. Chloride of tin added to a solution of corrosive sublimate, produces at first a white and then a black precipitate which, after it has been boiled, is resolved into globules of metallic mercury easily separable by filtration. The stannous chloride should be strong and mixed with its volume of strong hydrochloric acid. If, while boiling, the mercurial compound is added to it, there is an immediate precipitation of metallic mercury. The same result is obtained with all the compounds of mercury excepting the sulphide. 2. Sulphuretted hydrogen and sulphide of ammonium produce, after a time, a black sulphide, not soluble in alkalies or diluted acids. 3. If the liquid is acidulated with hydrochloric acid and bright copper foil, wire, or gauze is plunged into it, the copper will soon acquire a silvery-white deposit, even in the cold, but more rapidly by heat. When the copper with the metallic deposit is heated in a tube, globules of mercury are obtained. (Figs. 30 and 31.) The chlorine with which the mercury is combined in the solution may be detected by nitrate of silver.

FIG. 28.



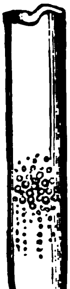
Prismatic crystals of corrosive sublimate from solution in water, magnified 30 diameters.

FIG. 29.



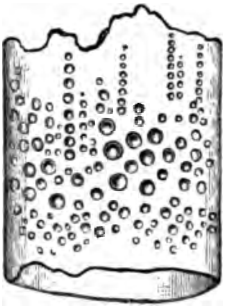
Crystals of corrosive sublimate from a solution in alcohol, magnified 80 diameters.

FIG. 30.



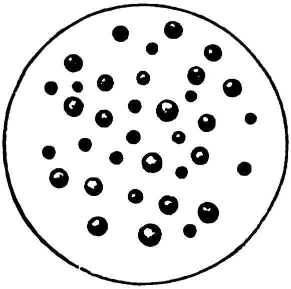
Mercury sublimed in globules, in a reduction-tube.

FIG. 31.



The same, magnified.

FIG. 32.



A small sublimate of mercury, magnified 124 diameters.

In Organic Liquids.—Corrosive sublimate may be sometimes obtained by decantation as a heavy sediment from the mucous and bloody contents of the stomach or the matters vomited. These should be separated, dried, and weighed. Unlike arsenic, corrosive sublimate in solution is precipitated as an insoluble compound by many organic principles, such as albumen, fibrin, casein, mucous membrane, also by gluten, tannic acid, and other vegetable substances. Thus, then, we must not expect to find it in all cases in a state of solution in the stomach. After removing the mineral sediment, we may separate any of these insoluble compounds, reserving them for further analysis.

As a trial-test for the presence of mercury *in solution* in the organic liquid, we may employ copper gauze or foil. A small portion of liquid acidulated with one-fourth part of hydrochloric acid, is brought to the boiling-point, and a small piece of gauze at the end of a fine platinum wire, introduced into the acid liquid. The copper may acquire after a short immersion, a white, gray, or silvery tarnish. In cases in which the quantity is small, the deposit does not take place until after the lapse of some hours. The copper should be removed, washed in water and alcohol, and dried and examined by a low power of the microscope. The deposit of any white metal on the copper will then be perceptible. It may be rolled into a cylinder and heated in a dry reduction-tube, when minute globules will appear as a sublimate. (See Fig. 32.) The sublimate in the tube should be examined with a microscope, and the copper should in all cases be heated, whether the red color of this metal appears to be covered or not by any deposit.

The metallic deposit, if any, as well as the globules, should be examined at once. Mercury is volatile at all temperatures, and in twenty-four hours a thin metallic film may disappear. I have known this to occur in a summer's day; while arsenic remains fixed on the metal for many years.

Reinsch found that one part of corrosive sublimate in 1000 parts of water, mixed with hydrochloric acid, gave immediately, to a small surface of copper, a white metallic film; in 5000 parts of water, without the addition of hydrochloric acid, there was no deposit in the cold, but the copper acquired a golden-yellow color on boiling. When boiled in a solution to which hydrochloric acid had been added it turned gray, becoming covered with a thin film of mercury, even when the quantity of water amounted to from 12,000 to 15,000 parts. (Gmelin's *Chemistry*, vol. 6, p. 57.) Although these experiments refer rather to the effect of dilution than the smallness of quantity detected, I entertain no doubt, from the results which I have obtained, that copper thus employed constitutes the most delicate test for separating mercury from corrosive sublimate. A quantity of corrosive sublimate amounting to about the 144th part of a grain, diffused in 8000 times its weight of water, gave, in twenty-four hours, evidence of the presence of mercury. This appeared to me to be near the limits of the process for detecting mercury.

In the event of *mercury* being thus readily detected, we may next seek for corrosive sublimate. The viscid mucous liquid may be submitted to dialysis like arsenic (*ante*, p. 154). After a few hours a clear liquid will be thus obtained which may be concentrated by evaporation, filtered, and introduced into a stoppered tube. We then add to it its volume of pure ether, and agitate the liquid at intervals for half an hour. Allow the liquid to subside, pour off the ether into a dial-glass or watch-glass, and submit it to spontaneous evaporation. As the ether passes off the corrosive sublimate will be deposited in white silky-looking prisms. These may be purified by solution in water if necessary, and again crystallized. Corrosive sublimate may thus be separated from arsenic and other mineral poisons in solution. Its properties may then be determined by the application of tests to the solid or solution as already described (*ante*, p. 371). The crystals touched with a solution of iodide of potassium will produce a scarlet color.

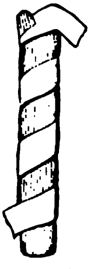
When the quantity of corrosive sublimate in solution is very small, ether may fail to separate it. In this case, the dialyzed liquid may be treated with the fluid tests as described at p. 371. Chloride of tin and nitrate of silver will give the reactions for mercury and chlorine, if any detectable quantity of the poison is dissolved in the organic liquid.

Let us suppose that the dialyzed liquid contains no trace of a mercurial compound, then there is no corrosive sublimate dissolved. We must then direct our attention to the analysis of the *insoluble matters* removed from the organic liquids. These may be boiled in distilled water, the liquid filtered, and tried by agitating it with its volume of ether. It will be found, when the analysis has not been long delayed, that most of the compounds which corrosive sublimate forms with organic matter yield commonly a sufficient quantity for detection by boiling them in water. Should water fail to extract the poison, the substance may be brought to dryness and heated with nitro-muriatic acid, until all the organic matter is decomposed, and the surplus nitric acid expelled. The residue may then be digested in water, and tested for mercury by the aid of copper gauze and hydrochloric acid (p. 372). This is also the process to be pursued with all organic *solids* supposed to contain the poison. The discovery of corrosive sublimate in small quantity in an organic liquid (medicine), in the matter vomited, or the contents of a stomach, does not necessarily prove that it has been administered as a poison, or with criminal intention. It is sometimes used medicinally; but only in solution, and in very small doses.

The galvanic process, or a combination of gold with zinc, has been frequently employed in place of copper for the separation of mercury from organic liquids. A layer of thin gold-foil is wound around a slip of zinc-foil in a spiral form, as represented in Fig. 33. This is suspended by a thread in the organic liquid, acidulated slightly with hydrochloric acid. It is allowed to remain immersed for some hours if necessary, in a warm place, or the liquid may be gently heated and set aside with the small galvanic pile.

If any soluble mercurial poison is present, even in small quantity, the gold will sooner or later lose its color and become whitened, while the zinc will be wholly or in part dissolved. The slip of gold-foil is washed in water and afterwards in ether, and dried. It should be divided into two equal portions. One should be submitted to heat in a tube, when globules of mercury will be obtained (Fig. 31); the other should be heated in a few drops of concentrated nitric acid until the gold has reacquired its yellow color. On evaporating the excess of nitric acid, and adding a solution of

FIG. 33.



A layer of gold-foil round zinc-foil.

chloride of tin, a dark gray precipitate of metallic mercury is thrown down.

It may be remarked that sublimed mercury is wholly unlike any other volatile substance. The perfect sphericity of the globules, their silvery whiteness by reflected, and complete opacity by transmitted, light, at once identify them as metallic mercury. This sublimate differs from that of arsenic in the fact that, when heated, it is resublimed in globules without change. It is not oxidized (like metallic arsenic), by heating it in a reduction-tube, but is simply transferred with its metallic lustre from one part of the tube to another.

The yellow color of the gold may not have been concealed by the mercurial deposit, owing perhaps to its great tenuity. Hence it is always proper to heat the gold in a reduction-tube before coming to the conclusion that mercury is really absent. The tube itself may not show a sublimate to the naked eye, owing to the minuteness of the globules, and to the fact of their being much scattered. In all cases it should be examined, at first with a low, and subsequently with a high power of the microscope. Minute strings of globules, varying from the 8000th to the 16,000th of an inch in diameter, may thus be detected. They are frequently deposited in a kind of chain in any minute crack or line on the interior of the glass reduction-tube. (See Fig. 31, p. 372). In the event of a doubt existing respecting the mercurial nature of the sublimate, the following experiment will remove it. Cut off with a file the portion of glass on which they are deposited; introduce this into a wide short tube, with a few drops of hydrochloric and half the quantity of nitric acid. Heat the acid liquid, and carry it to dryness on a sand-bath. White prismatic crystals of corrosive sublimate will remain, if the sublimate was of a mercurial nature, and too great a heat has not been applied. On touching the white residue cautiously with a drop of solution of iodide of potassium, the crystals will acquire a scarlet-red color.

Another method may be adopted. Place the suspected organic liquid in a small golden capsule. Acidulate it slightly with hydrochloric acid, and touch the gold, through the acid liquid, with a slip of pure zinc-foil. Mercury will be deposited in a white silvery stain on the gold, wherever the two metals have come into contact. Wash out the capsule with distilled water, and add a few drops of strong nitric acid. Pernitrate of mercury is thus obtained, which may be tested by the processes required for the detection of the persalts of mercury (p. 389). Dilution interferes with this process, hence it is desirable to concentrate the liquid as much as possible. One-sixteenth of a grain of corrosive sublimate, dissolved into sixteen ounces of water, gave no deposit on gold-foil with zinc; but when the quantity was dissolved in one ounce of the organic liquid, metallic mercury was separated, and its properties demonstrated in less than half an hour.

Absorbed and eliminated Mercury.—Although absorbed mercury, like other metals, is eliminated from the system, yet its elimination through the ordinary secretions appears to be slow, and subject to some uncertainty with respect to the time at which it commences, as well as to the duration of the process.

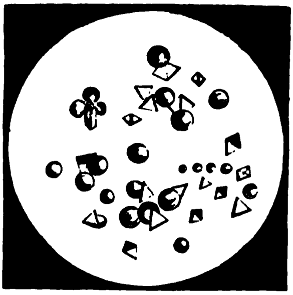
In the search for mercury in living persons laboring under the effects of this poison, a large quantity of urine should be examined, and an analysis made at intervals. The urine should be evaporated to dryness, and the dry residue or extract treated by the following process, which is the same as that required for the *tissues*. From four to eight ounces of the liver or other organs should be dried, broken up, and then boiled until dissolved, in one part of pure hydrochloric acid and four parts of water. The acid liquid may be strained through linen, and the residue pressed. The liquid, if in large quantity, should now be concentrated by gentle evaporation, and while still warm, treated with copper in the manner already described (*ante* p. 372).

Most of the common medicinal preparations of mercury are in an insoluble form. So far as the tissues are concerned, the chemical result would be the same whether the mercury was taken in a dissolved or undissolved state.

The efficiency of this method of detecting mercury when absorbed and deposited in the tissues, is indicated by the following fact. In an alleged case of poisoning (May, 1864) a child died after an illness of twenty-two hours. Fourteen hours before death, two grains of calomel had been given to it. This had caused much purging, and mercury was found in one of the last evacuations passed. Four ounces of the liver were treated with hydrochloric acid and water, and a small piece of pure copper placed in the acid liquid while warm, and kept there for about forty-eight hours. It acquired a slight silvery lustre, and globules of mercury were obtained from it by sublimation.

If arsenic should be present in the tissues at the same time, and the acid mixture is boiled, arsenic and mercury will be deposited together; and when the copper is heated, the globules of mercury will be obtained nucleated or intermixed with octahedral crystals of arsenious acid (Fig. 34). In a case of exhumation after twenty-one months' burial, these mixed sublimates were obtained by the examination of the rectum of the deceased. (*Reg.* v. *Bacon*, Lincoln Summer Assizes, 1857.) It appeared in evidence that arsenic had been administered to the woman a day or two before her death, and a dose of calomel had been prescribed more recently. This accounted for the presence of the mixed sublimates. When mercury and arsenic are thus associated in organic liquids, the arsenic may be entirely separated from mercury by the distillation process (*ante*, p. 319).

FIG. 34.



Mixed sublimates of mercury and arsenious acid, magnified.

In the living body, mercury is eliminated by the saliva as well as by the urine. About one drachm of this fluid will suffice for the detection of the metal by the following process. Acidulate the saliva with one-fourth of its volume of pure hydrochloric acid. Immerse in this a portion of copper gauze, about the sixteenth of an inch square, attached to a fine platinum wire. Place the tube containing the liquid in a warm place for a few hours. If mercury is present in the saliva, the copper gauze will be whitened. Other portions may be then introduced until the mercury ceases to be deposited. The pieces of copper should be washed in water and ether, dried, and examined by a low power of the microscope, and then heated in a small reduction-tube. Globules of mercury visible under the microscope will then be obtained. In a case of inunction with mercury the metal was thus detected in the saliva on the third day. There was painful swelling of the salivary glands, with the peculiar metallic taste produced by mercury. This analysis of the saliva may not only furnish evidence that the patient is under the influence of mercurial poison, but it will prove, in a case otherwise doubtful, whether the *salivation* from which a person is suffering is owing to mercury or some other cause. An examination of the saliva should be made in other cases of metallic poisoning, as arsenic, antimony, and other metals might be thus detected in the act of elimination from the living body. As mercury is not a constituent of the human body, the discovery of it in the tissues proves that it must have been received *ab extra.*

By one or other of the processes above mentioned we may be able to show the presence of *mercury*, but not of corrosive sublimate, in the body. Whether the mercurial compound had acted as a poison or not, must be determined from symptoms and appearances; whether it had been given or taken as a medicine or not, is a conclusion which must also be determined from other circumstances. The proof that the mercury was really in the form of corrosive sublimate could only be derived from the discovery of some undissolved portions of the solid poison in the stomach or its contents, or from an actual separation of the poison itself by means of ether. If thus obtained after filtration of an organic liquid, it would show its presence in the form of a soluble salt: all the soluble salts of mercury are poisonous, and are rarely used internally as medicines. If undissolved, the absorbed mercury may have been derived from some mercurial medicine innocently taken by the deceased. Nothing is more common than to discover traces of mercury in the stomach, bowels, liver, kidneys, or other organs of a dead body. No importance can be attached to this discovery in the absence of evidence that the deceased has actually suffered from symptoms of mercurial poisoning. As to the mercury found in the tissues, it may have been derived from a soluble or insoluble medicinal compound, or from exposure to the vapors of the metal, or of its salts in various trades.

A person may die from the effects of corrosive sublimate, and none of the poison may be found in the stomach, and no mercury found in the tissues. A case of this kind occurred to me some years since at Guy's Hospital. Owing to corrosive sublimate readily combining with the mucous membrane, it is more likely to be detected than arsenic. In a well-marked case, which occurred to Mr. Watson, where two drachms killed a person in six days, none was found on a chemical analysis of the contents. In a case in which two drachms were swallowed, and the man died in four days, no mercury was detected in the stomach or tissues; and in another, in which a similar quantity was taken, and death occurred on the *fourth* day, the stomach and tissues were examined by the galvanic gold test; but not a trace of mercury could be detected in them. A case occurred to Dr. Wegeler, in which a young man poisoned himself with three drachms of corrosive sublimate, and died on the *sixth* day; none of the poison could be detected in the stomach or intestines. (Canstatt's *Jahresbericht für* 1846, bd. v, p. 81; and Wharton and Stille's *Med. Jour.*, 1855, p. 440.) In Dr. Geoghegan's case, in which a large dose was taken, and the man died on the *fifteenth* day, the mouth, stomach, and intestines, as well as liver, spleen, and kidneys, were submitted to analysis; but there was no trace of mercury, either in the free or absorbed state. (*Reg.* v. *Walsh*, *Med. Gaz.*, 1850, vol. 46, p. 255.) Referring to what has elsewhere been said on the elimination of mercury, these facts corroborate the view of M. L. Orfila—namely, that in acute poisoning by this mineral, if the person survives *fifteen* days, it is probable that no trace of the metal will be found in the body. "The experts will, however," he remarks, "commit a gross error if they conclude from this that there has been no poisoning." (*Comptes Rendus*, Jan. 15th, 1852; and Wharton and Stillé, *Med. Jour.*, p. 441.)

Quantitative Analysis.—If the poison be entirely in a soluble form, we may procure the corrosive sublimate from a measured portion of liquid by the use of ether, and calculate the remainder proportionably. If it be in an insoluble form, we must then pursue the process elsewhere described, and precipitate it entirely by chloride of tin, purifying the mercury by boiling it first in potash, and secondly in hydrochloric acid. For every 100 grains of metallic mercury obtained, we must allow 135 grains of crystallized corrosive sublimate to have been present. Lassaigne advises the use of sulphuretted hydrogen. (*Ann. d'Hyg.*, Juillet, 1858, p. 204.)

CHAPTER XLI.

OTHER MERCURIAL COMPOUNDS—CALOMEL—WHITE PRECIPITATE OR AMMONIATED MERCURY—RED PRECIPITATE—RED OXIDE OF MERCURY—CINNABAR—VERMILION—CYANIDE OF MERCURY—TURPETH MINERAL—NITRATES OF MERCURY—MERCURIC METHIDE.

CALOMEL.

THIS substance, also called chloride or subchloride of mercury, although commonly regarded as a mild medicine, is capable of destroying life, even in comparatively small doses. Several cases have been already referred to, in which excessive salivation, gangrene of the salivary organs, and death have followed as a result of idiosyncrasy from the medicinal dose of a few grains (p. 356). There is a case reported in the *Medical Gazette* (vol. 18, p. 484), in which a boy, æt. 14, was killed in about three weeks by a dose of only *six grains* of calomel. It is singular that in this case neither the teeth nor the salivary glands were affected; still, considering the effects of calomel in other instances, it seems most probable that the ulceration and gangrene of the face, which followed, were due to it. Pereira mentions the case of a lady who was killed by a dose of twenty grains of calomel. She had previously taken a moderate dose without a sufficient effect being produced. Sobernheim states that a girl, æt. 11, took in twenty-four hours, eight grains of calomel for an attack of croup, and died in eight days from inflammation and ulceration of the mouth and throat. In another instance, which occurred to Lesser, fifteen grains of calomel produced similar effects, with excessive salivation; and this patient also died in eight days. Meckel relates that twelve grains have destroyed life. (*Lehrbuch der Ger. Med.*, p. 267.) Two cases of death from calomel, in children, are recorded in the *Registration Returns* for 1840.

There are many other fatal cases on record, and the facts seem to leave no doubt that calomel may, in large doses, act as an irritant poison. It was supposed that these effects might be ascribed to the calomel being adulterated with corrosive sublimate; but this supposition is not well founded. It has also been suggested that calomel might be converted into corrosive sublimate by the free hydrochloric acid present in the stomach; but the small proportion in which this acid exists is adverse to this suggestion. (*Ed. Med. and Surg. Jour.*, vol. 49, p. 336.)

Analysis.—1. Calomel is insoluble in water, alcohol, and ether. By either of the latter liquids corrosive sublimate may be detected and separated from it. 2. When heated on platinum or mica, it is entirely volatilized. 3. Potash and ammonia render it black. 4. Heated with dry carbonate of soda, it yields a sublimate of metallic mercury in globules, and leaves a white residue of chloride of sodium, which may be examined by the same process as the residue of corrosive sublimate (*ante*, p. 372). It is instantaneously reduced to the state of metallic mercury when added to a boiling solution of chloride of tin in hydrochloric acid. Owing to its great weight and insolubility, calomel may be easily separated from *organic liquids* and solids by simply washing them and decanting the water.

WHITE PRECIPITATE—AMMONIATED MERCURY.

This is an irritant mercurial compound, which is much used by the poor as a local application for ringworm and other skin diseases. A few years since it was a contested question whether it was or was not a poison; and at the Chelmsford Lent Assizes in 1850, a woman, who was indicted for administering this substance to her husband, owed her acquittal to the lenient but erroneous assumption in her favor that it was *not* a poison. Out of fourteen cases which I have collected, in which white precipitate was taken in doses varying from a few grains to forty, two only proved fatal.

Dr. Pavy's experiments on dogs and rabbits show that this is a more formidable poison than it has been hitherto supposed to be. The greater number of recoveries have been probably owing to the substance being early ejected by vomiting. Rabbits, which do not vomit, were killed by a dose of four or five grains in a few hours. After death, mercury was found deposited in various organs, but more in the kidneys than in the other viscera. (For additional facts connected with the action of this poison, see *Guy's Hosp. Rep.*, October, 1860, p. 483.)

Symptoms and Appearances.—The symptoms which it produces are violent vomiting, cramps, purging, and griping pain in the stomach, with convulsions. After death there is more or less inflammation of the stomach and bowels. In August, 1863, a woman, æt. 30, swallowed a pennyworth of white precipitate, and shortly afterwards a pennyworth of acetate of lead. In half an hour there was violent vomiting with pain over the whole of the abdomen. An hour and a half afterwards the symptoms were, in addition to the abdominal pain, great thirst and a comatose condition. On the second day, there was slight tenderness of the gums, a flow of saliva, flushed face, with great tenderness of the abdomen; and on the fourth day profuse salivation. This subsided on the tenth day, and the woman recovered. (*Med. Times and Gazette*, 1863, pp. 2, 645.) A young woman swallowed about thirty or forty grains of this substance by mistake for carbonate of soda. The chief symptoms were pain in the stomach and a spasmodic twitching of the muscles of the left arm and leg. These spasms continued for twenty-four hours. Emetics were given, and she recovered. (*Lancet*, 1871, vol. 2, p. 540.)

Salivation is one of the symptoms when the person survives. In the case of a child this substance caused death by exhaustion as a result of excessive salivation. In the following case, reported by Dr. Stevenson, it was a prominent symptom and was an accelerating cause of death. An adult woman was admitted into Guy's Hospital in 1872, suffering from profuse salivation, and she died in about a week after taking an unknown quantity of white precipitate by mistake for magnesia. The chief symptoms on her admission were excessive salivation, fetor of the breath, sloughing of the gums, and death from exhaustion. At this time there were no symptoms of irritation in the stomach and bowels. On inspection, the gums were found eroded, the alveoli exposed, and some of the teeth had dropped out. The stomach was ecchymosed in patches, corrugated, and in a state of post-mortem solution. There was also an hour-glass contraction. The small intestines were of a pink color, and at the lower end dark and slaty colored. The cæcum was red and injected, and it as well as the colon had a slaty color. The other organs presented nothing remarkable. (*Guy's Hosp. Rep.*, 1874, p. 415.)

Dr. Proctor, of York, communicated to me a case in which a woman recovered after having taken *forty grains* of this substance. In half an hour she complained of pain in the throat extending to the stomach, and her mouth was dry and clammy; the mucous membrane of the lips and tongue was slightly blistered. There was neither vomiting nor purging until a dose of tartar emetic and castor-oil had been given. In about three days she recovered. A case occurred to Mr. Michael, of Swansea, in which a woman, æt. 37, swallowed not less than *one hundred grains* of white precipitate. She was seen in three hours and a half; she complained of great pain in the stomach, with cramps of the left side and lower limbs, coming on at intervals of two or three minutes. The pulse was rapid, weak and thready, the surface cold and clammy, and the tongue red. She had vomited a thick white tenacious mucus with a white sediment; vomiting was kept up for hours. The bowels were freely opened. There was great prostration, continuing for several hours, and the pain in the stomach remained for three or four days, after which it gradually subsided. (*Brit. Med. Journal*, Oct. 31, 1857, p. 909.) In these two cases there was no salivation, but in one which occurred in the practice of Mr. Giles, this was a prominent symptom. A girl swallowed about half a drachm of white precipitate in a cup of tea. She suffered much pain in the stomach, and there was frequent purging. On the following day there was swelling of the face and gums, with salivation, which lasted several days. She recovered in about nine days. (*Lancet*, 1857, vol. 2, p. 9.) Judging from these cases, white precipitate cannot be regarded as an active poison; and its effects are somewhat uncertain. It must, however, be ranked among mercurial poisons.

There have been several convictions for poisoning or the attempt to poison with this substance. At the Exeter Lent Assizes, 1855 (*Reg.* v. *Daniel*), a boy was convicted of the attempt to administer poison to his father. A trial for attempting to poison by this compound took place at the Maidstone Summer Assizes, 1869 (*Reg.* v. *Seaham*). The compound is white, but, as a result of boiling, it gave a yellow color to the gruel in which it was administered. This is a remarkable property which would attract the attention of persons not acquainted with chemistry. It has sufficed to create suspicion of poisoning in more than one case. In February, 1873, a boy of 12 was convicted, at the Central Criminal Court, of administering this poison feloniously in medicine. The prosecutor experienced a hot sensation, unlike the bitter taste he had before perceived. A white powder was found floating which proved to be white precipitate. In *Reg.* v. *Hargreaves* (Manchester Lent Assizes, 1866), a girl was convicted of an attempt to poison her father by this substance. The poison was put into milk and medicine. It produced a burning sensation in the throat and stomach, and thus led to suspicion. About ten grains of white precipitate were detected in some buttermilk. A trial has recently taken place involving a charge of poisoning with white precipitate mixed with condensed milk (*Reg.* v. *Clapp*, C. C. C., June, 1874). The milk was taken by several persons and caused violent sickness with other symptoms of mercurial poisoning. They all recovered. The prisoner was convicted and sentenced to seven years' penal servitude.

In *Reg.* v. *Moore* (Lewes Lent. Ass., 1860), a woman was charged with killing her infant with white precipitate. This case presented some remarkable features, and the late Dr. Miller, Dr. Pavy, and myself were consulted with respect to it. The infant was three months of age. It was admitted with the mother into the workhouse on Nov. 17, and died on Dec. 21. The child had suffered from disorder of the bowels; and gray powder had been prescribed for it. Of this it had taken, in divided doses, about eight grains up to within four days of its death. On the night of Dec. 20, it was attacked with violent vomiting and purging and severe pain in the abdomen. It died in forty-three hours after the commencement of this severe attack. It was proved that shortly before the symptoms became thus aggravated, the prisoner had procured a pennyworth of white precipitate. She gave a false reason for the purchase, and none of the powder could be found. On examining the body the principal appearances were an inflammatory redness of the mucous membrane of the œsophagus, stomach, and small intestines. A white insoluble substance, having all the properties of white precipitate, was found in the stomach. This was mixed with mucus and starchy matter. No gray powder was found, and no chalk. Mercury was detected in the liver and kidneys, and in some mucous discharges on a napkin. The quantity of mercurial compound present in the parts examined was estimated at from three to five grains.

The previous illness of the infant, coupled with the fact that it had taken eight grains of gray powder, led to a doubt whether death could be strictly assigned to white precipitate. The mercury found in the liver and kidneys might have been owing to the gray powder taken. It was proved that white precipitate had been administered; and the woman was convicted on a charge of administering poison with intent to murder. The specific symptom of salivation was wanting in this case, but had this symptom existed it might still have been ascribed to the gray powder. The amount of white precipitate given was unknown. As it was sold it was adulterated with starch, and contained corrosive sublimate. There could be no doubt that the death of this infant was either caused or accelerated by white precipitate, but owing to the long previous illness and the fact that mercury had been prescribed for it, the authorities withdrew the indictment for murder. For further details the reader is referred to the *Guy's Hosp. Reports*, 1860, p. 483.

White precipitate is not used internally. In the British Pharmacopœia it is directed to be employed only as an ointment, the proportion being one part in eight. It is easily procured by children. It is sold to the public at the rate of one penny for a scruple or a drachm.

This compound may cause mercurial poisoning by absorption as a result of external application, especially when it contains corrosive sublimate. I have not met with any fatal case, but in one instance, in which it was mixed with arsenic as an ointment, and applied to the scalp, it caused the death of a child, and led to a strong suspicion of murder. (See the case of Bootman, *Guy's Hosp. Reports*, 1864, p. 220.)

Analysis.—White precipitate is a heavy, chalky-looking substance seen in powder or lumps. It contains about eighty per cent. of mercury. As it is sold in the shops, it frequently contains corrosive sublimate (from which it is made) to the amount of one or two per cent. It is quite insoluble in water, alcohol, and ether. Owing to its insolubility and great weight, it may be separated from the liquid contents of the stomach by washing and decantation.

When boiled in water it acquires a yellow color from partial decomposition. It is soluble in acids (nitric), and is not, like calomel, blackened by alkalies. The nitric acid solution gives a precipitate with nitrate of silver, showing the presence of chlorine. This acid has no solvent action on calomel. When boiled with a solution of potash it yields ammonia, and yellow oxide of mercury is deposited. When heated in the dry state with carbonate of soda it evolves ammonia, and gives a sublimate of metallic mercury in globules. When added to a boiling acid solution of chloride of tin it is instantly reduced to metallic mercury.

Corrosive sublimate may be detected in it by digesting the powder in ether, and filtering and evaporating the ethereal liquid.

The mercury derived from this compound and deposited in the tissues may be detected by Reinsch's process. (See *ante*, p. 373.)

RED PRECIPITATE—RED OXIDE OF MERCURY.

Symptoms and Effects.—This substance is poisonous, but instances of poisoning by it are very rare. The following case occurred at Guy's Hospital in 1833. A woman, æt. 22, who had swallowed a quantity of red precipitate, was brought in laboring under the following symptoms: The surface was cold and clammy, there was stupor approaching to narcotism—frothy discharge from the mouth, and occasional vomiting; the vomited matters contained some red powder, which was proved to be red precipitate. There was considerable pain in the abdomen, increased by pressure; and there were cramps in the lower limbs. On the following day the mouth and throat were painful, and the woman complained of a coppery taste. The treatment consisted in the use of the stomach-pump and the free administration of albumen with gluten. She left the hospital in four days, still under the influence of mercury. The quantity of oxide here taken was not ascertained.

Mr. Alison met with a case in which thirty-five grains were taken. Emetics were given, and the stomach-pump was used. The woman gradually recovered, having suffered from a burning pain in the stomach. (*Lancet*, N. S., vol. 19, p. 401.) Sobernheim relates a case in which a man, æt. 26, swallowed an ounce of red precipitate. He was speedily attacked with pain in the abdomen, nausea, purging, cramps, and general weakness. The vomited matters consisted of masses of mucus containing red precipitate. He continued to get worse, and died in less than forty-eight hours after taking the poison. On inspection, the mucous membrane was found eroded and inflamed in patches, small particles of the poison being imbedded in it. The duodenum was in a similar state, and there was a large quantity of red precipitate in the contents of this intestine, as well as in the stomach. (*Op. cit.*, 250.)

A girl, æt. 15, took by mistake half an ounce of red precipitate. She was admitted into the hospital a few minutes afterwards, suffering from no urgent symptoms. An emetic of sulphate of zinc was administered, and an abundance of milk ordered. The following day her lips, gums, and mouth were very sore, swollen and reddened. There was mercurial fetor in the breath, with headache, and pain in the region of the stomach. She was ordered an opiate mixture and castor-oil. In about a week she lost two front teeth, and her mouth remained slightly sore for a few days longer. She ultimately recovered. (*Brit. Med. Jour.*, Jan. 10, 1874, p. 40.)

This compound is a poison to animals. In July, 1874, an action was brought against a druggist for the loss of a dog. The owner had sent for turpeth mineral, and the druggist supplied the red oxide of mercury. He gave the animal from thirty to forty grains, and it suffered great pain, and died the following day. A verdict was returned for the plaintiff. (*Pharm. Jour.*, July, 1874, p. 76.)

A common opinion exists among the vulgar, that red precipitate is possessed of very active poisonous properties; hence it is sometimes administered with criminal design.

Analysis.—Red precipitate is known—1. By its being in red crystalline scales. 2. By its insolubility in water—this, together with its great weight, renders it easy of separation from *organic liquids*. 3. It is readily dissolved by warm hydrochloric acid, forming a solution possessing all the chemical properties of a solution of corrosive sublimate. (See *ante*, p. 372.) 4. When heated in a small tube, it becomes black (reacquiring its red color on cooling); and, while an abundant sublimate of mercury is formed, oxygen gas is evolved. Red precipitate is sometimes adulterated with red lead.

CINNABAR—VERMILION—PERSULPHIDE OF MERCURY.

The term *Cinnabar* is applied to a dark and heavy compound of sulphur and mercury, while *Vermilion* is the same substance reduced to a fine powder. It is well known as a red pigment, and is often employed in coloring confectionery and wafers. I have not been able to find any instance of its having acted as a poison on man. Orfila believes that it is not poisonous. It has, however, proved fatal to animals in the proportion of from thirty to seventy grains, even when applied externally to a wound. Cinnabar is sometimes used for giving a red color to ointments, *e. g.*, the sulphur ointment. In such cases the quantity used is very small, and can do no injury even if swallowed. This substance is also sometimes employed by dentists as a coloring matter to vulcanized rubber or gutta-percha for mounting artificial teeth. Although it cannot be regarded as an active irritant poison in the stomach, the placing of it in such a situation that it would be always in contact with the mucous fluids of the mouth, is liable to lead to the usual consequences of chronic poisoning by mercury. In May 1854, a medical man consulted me under the following circumstances. Upon the recommendation of a dentist, he had worn this red composition as a frame for false teeth, in place of gold. After some time he perceived a metallic taste in his mouth, the gums became inflamed and ulcerated, there was great weakness and want of nervous power, with pains in the loins and an eruption on the legs. When the composition was removed, these symptoms abated. I examined the substance, and found in it a great quantity of vermilion; it had been mixed with the sulphur and rubber to give the appearance of the red color of the gums. Dr. Wells, of Reading, has directed the attention of professional men to accidents of this nature. A patient of his, who had been provided with a frame of this description for the upper and lower jaws, perceived, soon after wearing it, a metallic taste in his mouth. His health failed, he lost his appetite, and became emaciated; he suffered from flatulency, fetid breath, and looseness of the bowels; his pulse was 100 and weak, and his tongue coated with a white film. This gentleman was peculiarly sensitive to the action of mercury. He left off wearing the teeth, and became gradually better and stronger. (*British Med. Jour.*, Sept. 5, 1863, p. 366.)

Other facts of a similar kind have been observed by Dr. B. Woodman. He found the pink and red vulcanite used for artificial gums or palates, to derive their color from vermilion in variable proportions. Some consisted only of blanched gutta-percha with vermilion. These plates slowly lose the mercurial compound in contact with the saliva. Dr. Woodman has found salivation and other indications of mercurial poisoning to result when these colored plates have been used, and these symptoms have only subsided on removing them. (*Pharm. Jour.*, Dec. 19, 1874, p. 485.)

Dr. Sutro has published a short abstract of a case in which the *vapor of vermilion* applied externally produced severe symptoms. A woman, by the advice of a quack, applied this vapor to a cancerous breast. She employed three drachms of vermilion, covering herself with a sheet, so that the vapor should only reach the body externally. After three fumigations, she suffered from severe salivation and violent fever, which continued for four weeks. The right arm became œdematous. (*Medical Times*, Sept. 27, 1845, p. 27.)

CYANIDE OF MERCURY.

Symptoms and Effects.—This is a substance which is but little known except to chemists, yet it is an active poison, and has caused death in at least two instances. As a mercurial poison it is not much inferior in activity to corrosive sublimate, but it has no corrosive properties. In April, 1823, a person who had swallowed *twenty grains* of this compound (thirteen decigrammes), was immediately seized with all the symptoms of poisoning by corrosive sublimate, and died in nine days. There was continued vomiting, with excessive salivation, ulceration of the mouth and fauces, suppression of urine, purging, and lastly convulsions of the limbs. On inspection, the mucous membrane of the stomach and intestinal canal was extensively inflamed. (*Orfila*, vol. 1, p. 583.) Sir R. Christison quotes a case in which *ten grains* destroyed life within the same period of time (*On Poisons*, p. 427); and in this case the symptoms were severe irritation of the stomach, inflammation of the mouth, and suppression of urine. These facts are adverse to the theory of Bernard, namely, that the cyanide when swallowed acts as a poison by reason of the production of prussic acid as a result of the action of the acid secretions of the stomach upon it. (*Sur les Substances Toxiques*, Paris, 1857, pp. 66, 103.) He carries this view so far that he believes if, in an animal out of health, the gastric juice were not secreted in its normal state of acidity, the cyanide would exert no poisonous action on the body! As a poison, the cyanide is probably not much inferior in activity to corrosive sublimate, but it differs from this compound in not possessing any locally corrosive action. There is a compound called *sulphocyanide of mercury*, which, when burned, gives obnoxious fumes of the metal.

Analysis.—When heated in a reduction-tube it yields cyanogen, a gas which burns with a rose-red flame and a blue halo, and metallic mercury is at the same time sublimed in globules. The cyanide is soluble in water. The solution differs from that of corrosive sublimate in not being precipitated by potash. Mercury is readily obtained from it by deposition on copper, and, when the cyanide is mixed with an acid, prussic acid is distilled over. It has no odor of prussic acid in solution.

TURPETH MINERAL—SUBSULPHATE OF MERCURY.

Symptoms and Appearances.—Fatal cases of poisoning by this compound are by no means common. Although insoluble in water, it is undoubtedly a strong irritant poison, and is capable of causing death in a comparatively small dose. A well-marked instance of its fatal operation was communicated to the Pathological Society by Mr. Ward, in March, 1847. A boy, æt. 16, swallowed *one drachm* of this preparation. It produced a burning sensation in the mouth and throat, and vomiting in ten minutes. In about an hour there was paleness, with anxiety of countenance, coldness of surface, constant sickness, sense of heat and constriction in the throat, and burning pain in the stomach with cramps. The irritability of the stomach continued in spite of treatment, and after two days there was salivation with mercurial fetor. The gums acquired a deep bluish tint and began to ulcerate. The patient died in about a week after he had taken the poison, without convulsions, and without suffering at any period from symptoms of cerebral disturbance. The principal *appearances* in the body were—inflammation of the gullet; its mucous membrane at the lower part peeling off; the inner surface of the stomach near the cardia and pylorus was covered with bloody spots; the small intestines were contracted, the inner coat was reddened, and petechial spots were found upon it, but chiefly in the large intestines. The parotid and submaxillary glands were swollen. Mercury was detected in the intestines. (See *Med. Gaz.*, vol. 39, p. 474.) From this account it will be perceived that turpeth mineral produces effects somewhat similar to those of corrosive sublimate, but it is less active.

Mr. Snoad, of Yoxall, has communicated to me the particulars of another case which was the subject of a trial for manslaughter at the Stafford Lent Assizes, 1862. A young man, æt. 27, by the mistake of a druggist, was supplied with turpeth mineral in place of Æthiop's mineral. He swallowed about two scruples of it, on an empty stomach, with a like quantity of cream of tartar and treacle. In ten minutes he was seized with violent vomiting and purging, the pulse was slow and small, the skin cold and clammy, and there was pain in the abdomen, especially in the region of the stomach. Under treatment, the symptoms of irritation abated, but never entirely subsided, and he died quietly on the eleventh day after taking the poisonous mixture. On inspection, the principal appearances were softening of the mucous membrane of the stomach and intestines, with patches of inflammation and dark discoloration. A small portion of the liver yielded mercury when treated with copper and hydrochloric acid. (*Guy's Hosp. Rep.*, 1864, p. 180.) The druggist who made this serious mistake was tried for manslaughter at the Stafford Local Assizes for 1862; but the jury considered that there had been no culpable negligence, and the prisoner was acquitted!

Analysis.—Turpeth mineral is a heavy powder of a yellow color, becoming of a dark olive by exposure to light. It is scarcely soluble in water, but has a strong metallic taste. When heated in a tube, with or without carbonate of soda, it yields metallic mercury. It may be analyzed by boiling it in potash, in which case sulphate of potash and peroxide of mercury result—the acid and the base are then easily determined. Mercury is immediately deposited from it on boiling it with a strongly acid solution of chloride of tin.

NITRATES OF MERCURY.

Symptoms and Effects.—These are corrosive poisons which are used for various purposes in the arts. They are solid white salts, soluble in cold water, if there be a little excess of acid present. The acid pernitrate caused death in a case reported by Mr. Bigsby, in the *Medical Gazette* (vol. 6, p. 329). A butcher's boy dissolved some mercury in strong nitric acid, and swallowed about a teaspoonful of the solution. Soon afterwards he suffered excruciating pain in the throat, gullet, and stomach—there was great anxiety, with cold skin, small pulse, colic, and purging. He became gradually weaker, and died in about two hours and a half. On inspection, the throat, gullet, and stomach were found corroded and inflamed. Although he survived so short a time, the mucous membrane of the stomach was of a deep red color.

M. Tardieu describes a case of poisoning by the acid nitrate of mercury which proved fatal in about the same time. A man swallowed a quantity of the acid nitrate in solution mixed with some hydrochloric acid. It produced immediately intense pain in the abdomen with violent vomiting—the liquids ejected causing effervescence on the pavement. In three-quarters of an hour he was in a state of collapse; face pale; lips of a violet hue; skin, especially of the limbs, cold and covered with sweat; pulse slow and irregular. He coughed up frothy blood, could scarcely speak, and the respiration was frequent and stertorous. He could not swallow, and liquids given to him were immediately ejected. There was purging with bloody feculent discharges soon after his admission. In spite of treatment, the breathing became more difficult, and the pulse imperceptible. He gradually sank, retaining his mental faculties until the last, and died two hours and a quarter after taking the poison. On inspection, the lining membrane of the mouth, throat, and œsophagus was found corroded and destroyed. The mucous membrane of the trachea and bronchi presented a dotted redness—that of the œsophagus in some parts was horny and the structures beneath were of a livid red color. The mucous membrane of the stomach was of a brick-red color, covered with a bloody mucous liquid. It was softened and pulpy at the greater end. The duodenum and the small intestines for some way downwards presented a similar appearance. (*L'Empoisonnement*, 1867, p. 231.)

I have elsewhere related a case in which the application of the pernitrate of mercury to the throat as an escharotic caused immediate death by asphyxia. (See *Guy's Hosp. Reports*, Oct. 1850, p. 206.) The acid nitrate of mercury has often been employed by accoucheurs as a local application in diseases of the neck of the uterus. In one instance in which it was thus used, the ordinary symptoms of mercurial poisoning showed themselves, and the patient appears to have suffered severely. (*Medical Gazette*, vol. 45, p. 1025.)

At the Leicester Summer Assizes, 1857 a girl was charged with administering nitrate of mercury to her mistress (*Reg.* v. *Smith*). The evidence showed that the accused had put the poison into some camomile tea prepared for the prosecutrix. Only a small quantity was taken, as the tea had a nauseous taste. The symptoms were: a burning sensation in the throat and stomach, violent vomiting, with severe pain in the abdomen. The woman recovered. In one case death took place under the usual symptoms from the external application of the nitrate in a liniment. (*Ed. Monthly Journal*, Aug. 1864, p. 167.)

A man, æt. 33, suffering from chronic poisoning by the nitrate of mercury, was admitted into Guy's Hospital on Dec. 10, 1863. He had been for four years engaged in packing the fur of rabbits, rats, and other animals, the dried skins of which had been previously brushed over with a solution of nitrate of mercury. For the first three years, he suffered only from a feeling of general weakness. About a twelvemonth since he could not hold his hand steadily enough to shave himself, and he soon afterwards lost completely all control over the voluntary movements of his limbs. Three or four months before his admission, he had had slight twitchings of his muscles when in bed. He was not at all emaciated. He said he had been salivated for about three months, soon after he began his occupation of packing furs; but his gums were not tender, and he had no metallic taste in his mouth. A month before his admission he gave up his work. When he became a patient under Dr. Rees I saw him, and his case was watched by Mr. Spurgin, one of my pupils. He could walk with assistance, but on standing or lying down he could not control his limbs, which trembled considerably. There were continued involuntry movements of his body and limbs, like those of chorea. He became much exhausted, owing to want of sleep; he perspired profusely. The urine was high-colored, but otherwise natural. Twelve ounces of it did not yield any mercury. No treatment appeared to give him rest or relief. Chloroform arrested the spasmodic movements, but only while he was under its influence. In five days he passed his urine involuntarily. He was more quiet, and slept a little at night. He had difficulty in swallowing; became gradually weaker, and died, apparently from exhaustion, on Dec. 24, a fortnight after his admission. On inspection, the body was found well nourished; the muscles were firm and healthy. The brain and spinal cord were carefully examined by Dr. Wilks, and were quite healthy. The lungs, heart, liver, spleen, and kidneys were free from any morbid appearance, or any change to indicate a cause of death. I made a chemical analysis of the brain, liver, and kidney. Six ounces of each organ were dried, and one-half of the dried residue, treated with hydrochloric acid and water, as elsewhere described, gave, in forty-eight hours, on a small portion of copper gauze, a grayish white deposit, which yielded globules of metallic mercury by heat. The kidney yielded the largest sublimate; but the quantity obtained from each organ was small, and might be described as in microscopical traces. The globules from the brain and liver had an average size of $\frac{1}{2800}$th of an inch; those from the kidney were larger. Dr. Whitley procured a portion of the fur similar to that which the man had been engaged in packing, and in a small quantity of this a soluble salt of mercury was readily detected. The case, which at first presented some difficulty in accounting for death, thus resolved itself into one of exhaustion as a result of chronic poisoning by mercury under somewhat unusual circumstances. It is probable that the man received the dust of the dried nitrate through the air which he breathed, as well as by contact with his mouth, nostrils, and skin. As other workpeople similarly engaged were not found to have suffered, this may have been a case of mercurial poisoning by idiosyncrasy. (*Guy's Hosp. Reports*, 1864, p. 173.) A report of other medico-legal cases will be found in the *Ann. d'Hyg.*, Juillet, 1842; and *Journal de Chimie*, 1846, p. 734.

Analysis.—In the solid state, the crystals, when heated in a tube, yield nitrous acid vapor—peroxide of mercury and globules of metallic mercury—when heated with carbonate of soda, metallic mercury is easily obtained. The *solution* possesses all the properties of corrosive sublimate, so far as the tests for mercury are concerned (*ante*, p. 373); but it gives no precipitate with nitrate of silver. When copper is immersed in it, mercury is deposited on the metal, and nitrate of copper is formed.

It may be observed of all solid mercurial compounds, that when heated in the dry state with anhydrous carbonate of soda, they yield sublimates of the metal in globules. All liquid and solid compounds give a dark precipitate of mercury when boiled with the acid chloride of tin.

MERCURIC METHIDE.

This is a heavy colorless liquid, containing 87 per cent. of mercury. It gives off a highly noxious vapor, producing the effects of mercurial poisoning in a more intense form than those observed among workmen exposed to the vapors of the metal.

Symptoms.—In February, 1865, a chemical assistant in the laboratory of St. Bartholomew's Hospital, who had been engaged for nearly three months in preparing mercuric methide, and who had been thus exposed to breathe the noxious vapors evolved in the process, was seized with dimness of sight, numbness of the hands, deafness, great weakness, swelling and tenderness of the gums; he moved his arms and legs with difficulty, and could not stand without support. In spite of treatment he became worse; an offensive odor issued from his breath and body, he was at times maniacal, and he died eleven days after his admission into the hospital.

Appearances.—The brain was congested, especially the gray matter, and there was congestion of the liver and kidneys. As none of the liquid had been swallowed, there was no appearance in the stomach and bowels calling for special notice. The symptoms indicated that death was owing to poisoning by the mercurial vapors of this compound. The case was left incomplete by the facts that no analysis of the tissues was made for the detection of absorbed mercury.

Another assistant, who had been exposed to the noxious vapors for a shorter time, suffered from similar symptoms. He had offensive breath, spongy gums, general impairment of the senses, and an affection of the brain, producing a state of idiocy. (*St. Bartholomew's Hospital Reports*, Oct. 1865; also *Chem. News*, Nov. 3, 1865, p. 213.)

CHAPTER XLII.

ON POISONING WITH LEAD—ACTION OF THE METAL—POISONOUS SALTS OF LEAD—THE ACETATE AND CARBONATE—SYMPTOMS OF ACUTE POISONING BY SUGAR OF LEAD AND GOULARD'S EXTRACT—EFFECTS PRODUCED BY CARBONATE OF LEAD—CHLORIDE, NITRATE, AND SULPHATE—CHRONIC POISONING—SYMPTOMS—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—PERIOD OF DEATH—TREATMENT OF ACUTE AND CHRONIC POISONING.

General Remarks.—Lead appears to exert no directly poisonous action when swallowed in the metallic state. Under exposure to air, water, and carbonic acid, it is easily converted into a poisonous white salt—carbonate of lead. In the interior of the body, however, we have no reason to believe that this metal produces any noxious effects. Bullets and shot are occasionally swallowed without giving rise to symptoms of poisoning. In a case that occurred to Dr. Davis, a boy, æt. 4, swallowed a leaden bullet. The child suffered no inconvenience after the bullet had reached the stomach. It was passed by the bowels in about a week, much roughened on the surface, and, by comparison with a bullet from the same mould, it had lost ten grains. (*Med. Gaz.* vol. 38, p. 686.) A man, æt. 23, swallowed in three days three ounces of small shot (No. 4). This is an alloy of lead and arsenic, the latter metal being in very small proportion. On the third day there was great anxiety and depression, with sunken features, coldness of skin, dizziness, and numbness in the arms and legs. He continued getting worse in spite of treatment; his bowels were obstinately torpid, and there was increased numbness in the arms and dizziness. Purgatives were given; the alvine discharges were examined, but only one pellet was found; so that if he had passed the shot at all, it must have happened in the three days before he was seen by his physician. This man perfectly recovered in a fortnight. (*Lancet*, Dec. 31, 1842.) As these are the symptoms produced by an oxide or a salt of lead, it is probable that the metal was partially oxidized and converted into a poisonous compound by the acid mucous secretions of the stomach. It was then absorbed, and produced its usual effects. The handling of metallic lead or pewter has been known to produce the effects of chronic poisoning. (See *ante*, p. 26.) In this case, probably, the lead is also oxidized; and when there is a want of cleanliness, it is brought to a state favorable for absorption by the chemical action of the secretions of the skin upon it. These contain chloride of sodium, which may convert the finely divided metal into chloride of lead in a state well fitted for absorption.

Cases are reported in which cattle and sheep have suffered from lead poisoning, and died under the following circumstances. In grazing, they have swallowed the thin splashes of lead left as a result of the volunteer firing at the butts. In some cases in which I was consulted in February, 1865, it appeared that in the bodies of three cows, two of which had died suddenly without any apparent disease, and the other had been killed, these splashings or flakes of lead were found in the stomachs. The lead adhered closely to the mucous membrane, which came off with the metal. The deaths took place so suddenly as to render it doubtful whether the lead could have been the cause. It seems that the two cows which died ate their food well the night before, and showed no sign of illness, but they were found dead the next morning. Under such circumstances, it is difficult to admit that there had been sufficient time for the oxidation and absorption of the lead. In the following cases there were clearly antecedent symptoms of lead-poisoning. Mr. Cox, of Hendon, who lost several sheep from this cause in 1865, observed that they first became very thin, and then paralyzed. When the bodies were examined, thin splashes of lead were found in them; and from one sheep he extracted half an ounce weight of lead, some pieces being as large as the finger-nail. He states that the splashes of metal were found within a semicircle of at least one hundred yards of the butts. The lead being in very thin flakes, presents a great surface, and is in a favorable condition for corrosion by the fluids of the stomach. This leads to rapid absorption.

Every salt of lead is poisonous, provided it is in a state fitted for absorption, either by the skin or the mucous membrane of the stomach. Dr. C. G. Mitscherlich found, by experiments on animals, that the acetate of lead mixed with acetic acid is more energetic than when given in the neutral state. (*Brit. and For. Med. Rev.*, No. 7, p. 208.) The only two salts of the metal which require special notice, in a medico-legal point of view, are sugar of lead and white lead.

SUGAR OF LEAD—ACETATE—SUBACETATE—GOULARD'S EXTRACT.

Symptoms—Acute Poisoning.—Acetate or sugar of lead is commonly met with in solid, heavy, crystalline masses, white, or of a brownish-white color. It resembles loaf-sugar in appearance, and has been mistaken for it. It is retailed to the public at the rate of from three halfpence to twopence an ounce; and for quantities less than this, one penny is charged.

Sugar of lead has a sweet taste, which is succeeded by an astringent or metallic taste. It is very soluble in water. Four parts of water at 60° will dissolve one part; and it is much more soluble at a boiling temperature. It is dissolved by alcohol.

This compound is by no means an active poison, although it is popularly considered to possess a virulent action. In medical practice, it has often been given in considerable doses without any serious effects resulting. Sir R. Christison states that he has given it medicinally, in divided doses, to the amount of eighteen grains daily for eight or ten days, without remarking any unpleasant symptom, except once or twice slight colic. (*Op. cit.*, 555.) When, however, the quantity taken has been from one to two ounces, the following symptoms have been observed: A burning, pricking sensation in the throat, with dryness and thirst; vomiting supervenes; there is uneasiness in the stomach, sometimes followed by violent colic. The abdomen is tense, and the parietes have been occasionally drawn in. The pain is relieved by pressure, and has intermissions. There is in general constipation of the bowels. If any fæces are passed, they are commonly of a dark color, indicative of the conversion of lead into sulphuret. The skin is cold, and there is great prostration of strength. When the case is protracted, the patient has been observed to suffer from cramp in the calves of the legs, pain in the insides of the thighs, numbness and sometimes paralysis of the extremities, with other symptoms of chronic poisoning. The affection of the nervous system is otherwise indicated by giddiness, stupor, and even coma. A well-marked blue line has been, in some cases, observed round the margin of the gums, where they join the teeth; but this is chiefly noticed when the case is protracted.

The symptoms of poisoning with this salt are subject to variation, but not to a greater degree than in other cases of irritant poisoning. Colic and constipation of the bowels are characteristic, and are generally met with; while the vomiting is commonly not very violent; it requires to be promoted by the use of emetics. A woman, æt. 41, was admitted into Guy's Hospital (May, 1846). It was ascertained that two hours previously she had swallowed about one ounce and a half, or half a teacupful of sugar of lead, dissolved in water. She experienced a nauseous metallic taste in her mouth, with a burning heat in the mouth, throat, and stomach. She took some water to remove the taste. This made her vomit. Her mouth was very dry; she had great pain at the pit of the stomach; and in two hours after the poison had been taken, she felt sleepy and stupid, alternately perspiring and shivering. She complained of a violent twisting pain in the abdomen, which was relieved by pressure; with this there was a sensation of sickness. She felt weak and languid; complained of cramp in the thighs, and numbness all over the body, with giddiness. The gums felt to the patient to be in lumps, and they were very tender; the breath was foul. The pulse was hurried, and the tongue coated; countenance anxious and excited; skin dry, cold, and hot alternately. The urine was passed freely. The next day there were pains all over the body, with numbness and sickness. On the third day she was very sleepy, but in less pain. For several days the abdomen was painful on the slightest pressure. She left the hospital in five days.

In March, 1858, a young man swallowed an ounce of acetate of lead in half a tumbler of water. He took what was undissolved as well as the portion dissolved. In a quarter of an hour he vomited. There was pain in the forehead, with severe pain in the abdomen; and in three-quarters of an hour from the time of swallowing the poison, he was purged once freely. When brought to the Northern Hospital, two hours subsequently, the only symptom was pain in the abdomen. The pulse presented nothing remarkable. The next day the bowels were constipated, and sulphate of magnesia was given. He was discharged well the day following. (*Med. Times and Gaz.*, March 20, 1858, p. 296.)

When the patient recovers from the first symptoms, the secondary effects often last for a considerable time. In two cases which occurred to Mr. Gorringe, two girls swallowed an ounce of the sugar of lead by mistake. Soon afterwards they felt a burning pain in the mouth, throat, and stomach, and in a quarter of an hour they vomited freely; in half an hour there was severe pain in the bowels, with purging. Under treatment, recovery took place. (*Prov. Med. Jour.*, April, 1846.) After a year had elapsed, they both suffered from severe pain in the stomach, which was tender on pressure. Nothing could be retained on the stomach; and there was a choking sensation in the throat, with some constitutional symptoms. A girl who had swallowed sixty grains of acetate of lead, and suffered severely from the primary symptoms, recovered and left the hospital in about three weeks, without any paralysis or other disorder affecting the muscular system. (*Lancet*, April 4, 1846, p. 384.) In another case, a woman, æt. 20, took one ounce of sugar of lead. She was placed under treatment at St. Thomas's Hospital. The chief symptoms were slight excoriation of the gums, a sensation of heat in the throat, and relaxation of the bowels. There was pain in the calves of the legs and thighs, and great thirst and restlessness. In six days the woman had quite recovered. (*Med. Gaz.*, vol. 5, p. 704.) Large doses given medicinally are not always borne with impunity. Dr. Joynes prescribed thirty grains of the acetate in four days for inveterate diarrhœa. The medicine appeared to be of service; but in a week afterwards the patient was seized with pain in the stomach; a severe attack of lead colic came on, and continued for eight days. (*Harrison on Lead Poison*, p. 148; see also *Provincial Transactions*, vol. 1, p. 119.)

The symptoms are sometimes slow in appearing. The following case occurred to Dr. Hviding. A girl swallowed about three drachms of the acetate of lead in broth. It was not until *two hours* afterwards that she began to experience sharp colicky pains in the abdomen, followed by vomiting. No medical treatment was employed for three days; and the only marked symptom then was obstinate constipation. Doses of castor oil were prescribed, and the girl recovered. (*Journal de Chimie*, 1845, p. 256.)

A series of cases of poisoning by acetate of lead has been reported by Mr. Bancks, of Stourbridge. (*Lancet*, May 5, 1849, p. 478.) By some accident, about thirty pounds of this substance were mixed at a miller's with eighty sacks of flour, and the whole was made into bread by the bakers, and supplied as usual to their customers. It seems that no fewer than 500 persons were attacked with symptoms of poisoning after partaking of this bread. In a few days they complained of a sense of constriction in the throat and at the pit of the stomach, violent crampy pains round the navel, rigidity of the abdominal muscles, a dragging pain in the loins, and cramp, with paralysis of the lower extremities. There was obstinate constipation, and the urine was scanty, and of a deep red color. The pulse generally was slow and feeble; the countenance anxious and sunken, frequently of a peculiar livid hue; tongue flabby; gums marked by a deep blue line. The surface was cool, and there was a general arrest of the secretions. Sickness was not a uniform symptom; and even when it existed at first, it speedily subsided. The mental faculties were undisturbed. Not one of the cases proved fatal; but among the more aggravated, there was great prostration, with collapse, livid countenance, universal cramps, numbness, and other alarming symptoms. After apparent convalescence, some of the symptoms returned in a more aggravated form, without any obvious cause, and for a long time the patients were out of health. Inflammation was not observed. Purgative medicines were found most effectual in the treatment. The quantity of acetate of lead taken by each person could not be determined, as, on analysis, the samples of bread were found to be very unequally impregnated with the poison.

Goulard's extract (*sub* or *tris-acetate of lead*) is generally seen under the form of a reddish-colored liquid, as it is often made with common vinegar instead of acetic acid. *Goulard water* is a lotion compounded of from one drachm to one drachm and a half of Goulard's extract, or solution of subacetate of lead, of a drachm of spirit, and a pint of water. It is not poisonous unless administered at intervals in small doses; it may then cause chronic poisoning. Goulard's extract, or subacetate of lead, has caused death in at least four instances—one in France and three in England. The symptoms produced are similar to those above described. In new-born infants this compound is likely to produce fatal effects. In a case in which a woman, who was suckling her infant, suffered from sore nipples, a wash of Goulard's extract was applied. The infant, eight days old, was put to the nipple, on which a coat of the lead-salt had dried. The result was, it suffered from the most severe colicky pains, and died on the eleventh day. (Bouchardat, *Ann. de Thérap.*, 1874, p. 188.)

The subacetate is much more powerful as a poison than the neutral acetate, probably from its containing a larger quantity of the oxide of lead. One fatal case of poisoning by Goulard's extract is recorded in the Coroners' Return for 1837–8. In January, 1840, two other cases of poisoning by it occurred in London in two children, aged respectively four and six years. The quantity taken by the children could not have been great, but they both died within thirty-six hours. The symptoms were at first violent vomiting and purging; in one case they resembled those of Asiatic cholera. Dr. Hall states that he gave ninety-x grains of this compound in three days—thirty-two grains in the rst four hours—and the only unpleasant effect was pain in the bowels. his was in a case of spitting of blood. (*Harrison on Lead Poison*, p. 45.) These results must not be considered paradoxical. They are, a great extent, explained by reference to the influence of disease on edicinal and poisonous agents. (See *ante*, p. 80.) Dr. Hall did not ke this quantity himself, nor try it on a healthy person, or the results ight have been different.

The CHLORIDE and NITRATE of lead are poisons, but not of an active ind. Dr. Christison found that it required 400 grains of the crystal- zed nitrate to kill a dog in sixteen hours. (*On Poisons*, p. 549.) ome years since, a woman, who had swallowed an unknown quantity f chloride of lead, was brought to Guy's Hospital. The only urgent ymptom was vomiting. She recovered and left the hospital on the ame day.

CARBONATE OF LEAD—WHITE LEAD.

Carbonate of lead, known also as *white lead*, *ceruse*, or *Kremser hite*, is commonly in the form of heavy white masses resembling halk. It is insoluble in water, but in large doses it possesses poison- us properties. In October, 1844, the late Dr. Snow met with the ollowing case of poisoning with this substance. A child, æt. 5, ate a ortion not so large as a marble, ground up with oil. For three days e merely suffered from pain in the abdomen and costiveness. On the hird night, the child became rapidly worse, and there was vomiting. e died ninety hours after taking the poison, having passed some ffensive motions of a greenish-black color (probably from sulphide of ad) before he died. It is remarkable that in this case so small a uantity should have proved fatal without exciting any marked symp- oms of irritation in the first instance.

There are many cases of poisoning by the carbonate of lead in the uman subject; but it has in these instances proved insidiously fatal, y inducing colica pictonum. The following case of recovery from a rge dose of carbonate of lead is reported by Mr. Cross. A woman, t. 33, took, by mistake for a dose of magnesia, from *six to eight rachms* of carbonate of lead. Five hours afterwards she was seen by er medical attendant. She was in a cold perspiration, breathing eavily, constantly vomiting, her pulse hard, small, and quick. There as great anxiety of countenance, with dryness of the throat; and a ense of heat in the stomach, with painful colics. Castor oil and sul- hate of magnesia, with diluted sulphuric acid, were given to her— he last at frequent intervals. The extensor muscles became paralyzed, nd the flexors rigidly contracted; the colics were so excruciating that he patient generally fainted after each paroxysm. The evacuations rom the bowels were of a dark color. The symptoms abated; but he next day there were nausea and faintness, with griping pains. In our days she was convalescent. A somewhat similar case is reported n Casper's *Wochenschrift* for 1844, in which a man, æt. 20, swallowed, y mistake for chalk, between five and six drachms of carbonate of ead. In a few hours it produced all the symptoms of irritant poisoning, thirst, burning pain, and incessant vomiting; yet, although he was not seen for twenty-four hours after taking the poison, he perfectly recovered in the course of a short time under very simple treatment. His recovery was probably due to the greater part of the carbonate having been ejected by the early vomiting. (*Ann. d'Hyg.*, 1845, vol. 2, p. 226.) These cases show that the carbonate of lead, although poisonous, is not very energetic. Its action as a poison is probably not greater than that of the acetate; and, so far as observations on the human subject extend, it is less active than the subacetate.

A case is related in the *Annales d'Hygiène* (April, 1844), which shows that shot used in cleaning bottles may be chemically acted on by the acids of the wine or liquid, and give rise to the production of this poisonous salt of lead. A person, after having swallowed a few glasses of liqueur, suffered from the most violent colicky pains, and all the symptoms of irritant poisoning. Dr. Hanle, who was immediately called, having observed that the liquor remaining in the bottle was very turbid, poured it off for analysis, when he found firmly wedged in, at the bottom of the bottle, ten leaden pellets, which had become so completely transformed into carbonate of lead, that there was only a small nucleus of the metal left. So long as the liquor was clear, no accident had arisen from its use; but the symptoms of poisoning appeared immediately when the turbid portion at the bottom of the bottle, containing the salt of lead either suspended or dissolved, was swallowed.

The *sulphate of lead*, by reason of its insolubility, is commonly regarded as inert, and the results of the following experiments appear to justify this opinion. M. Dupasquier ascertained that *seventy-seven* grains of the sulphate might be given to a dog, kept fasting for twenty-four hours, without exciting vomiting or any other unpleasant symptoms. The dog was kept for four days, and the dose produced no effect. On killing the animal and inspecting the body, there were no abnormal appearances. Doses of 150 and 300 grains were given to other dogs, without producing symptoms of poisoning. (Consult *Méd. Lég.*, 1843, p. 15.) Orfila states that he gave to a dog 554 grains in a finely pulverized state, without any injurious effects resulting. The dog ate its food as usual on the following day. (*Op. cit.*, vol. 1, p. 690.)

The *chromate* and *iodide of lead* are ranked among lead poisons. In reference to the *chromate*, the reader is referred to the compounds of chromium (*post*). It has been most improperly used as a yellow coloring for confectionery and ginger lozenges, in place of turmeric, and has thus given rise to accidents among children. With respect to the iodide, there is a single experiment on a cat recorded by M. Paton, from which we learn that nineteen grains in two doses produced paralysis of the hinder legs, and apparently colicky pains. The animal died in three days. (Orfila, *Toxicol.*, vol. 1, p. 702.)

OXIDES OF LEAD.

The yellow oxide (massicot), and the brown oxide (peroxide), are but little known except to chemists. *Litharge* and *Minium* or *red lead* are, however, much employed in the arts, and have sometimes given rise to accidental poisoning. In October, 1843, a woman who had swallowed two and a quarter ounces of the red oxide of lead, was admitted into Guy's Hospital. No symptoms appeared for nine hours. There was then colicky pain, with urgent vomiting, followed by headache and general tenderness of the abdomen. She entirely recovered in about twelve days. (*Guy's Hospital Reports*, October, 1850, p. 209.) In March, 1870, owing to an accident, some red lead, was mixed with a quantity of beer in a brewery at Guildford. Several persons who drank this beer suffered from lead-poisoning. One man died, but it was probable that disease of the lungs was the immediate cause of his death. Colicky pains and a blue line on the gums, with constipation, were well-marked symptoms among those who suffered. In the course of a month as many as twenty-seven cases of lead-colic came under treatment from this cause. (*Lancet*, 1870, vol. 1, pp. 428 and 495.)

Chronic Poisoning.—The effects of chronic poisoning are more frequently witnessed in reference to the salts of lead than of other metallic irritants. Any salt of lead taken in small doses at intervals, may give rise to chronic poisoning, producing lead-palsy or other forms of lead disease. White lead and litharge are the compounds to which chronic poisoning may be most frequently traced. The medicinal use of the acetate, if continued for an undue length of time, may so saturate the system with the metal as to occasion this form of poisoning. A child, æt. 6, took, in a quack medicine, 1-15th of a grain of acetate of lead two or three times a day for nearly nine weeks. It was then found to be laboring under symptoms of poisoning by lead, and two days afterwards the child died. The first effects of taking the medicine were that the child lost flesh, and complained of colicky pains; the bowels were constipated, the evacuations, when passed, black and offensive, and there was fetor of the breath. Latterly, the child was drowsy and the limbs were paralyzed. Upon the day of its death it was convulsed, and shortly before death it fell into a state of coma. (*Pharmaceutical Journal*, December, 1845, p. 259. Case by Dr. Letheby.) This case shows that more injury may be done by frequently repeated small doses than by one or two large doses. This child took in nine weeks, no more than Sir R. Christison prescribed, without any injurious effects, in the space of two days.

The disease called *Colica pictonum*, or *Painter's colic*, derives its name from its supposed seat in the colon, but according to some observers the seat of pain is in the muscular coverings of the abdomen. (Dr. Briquet, *Archives Générales*, Feb. and March, 1838; and *Dub. Hosp. Gazette*, Aug. 1858; p. 237.) This disease may be regarded as a *chronic* form of poisoning with carbonate of lead. The carbonate finds its way into the system, among white lead manufacturers, either through the skin or through the lungs, or both together; it is diffused through the air as a fine dust, and is not only respired, but taken into the mouth and swallowed with the saliva. It has been remarked that in factories where the powder was not in a dry state, not only have the laborers suffered, but horses, dogs, and even rats, have died from its effects. Since the practice has arisen of grinding the white lead in water, cases of colica pictonum have not been so numerous. They are still, however, not unfrequent among painters, plumbers, pewterers, the manufacturers of some kinds of glazed cards, the bleachers of Brussels lace, and among those engaged in the glazing of pottery, when oxide of lead is employed in the glaze. In ten years, according to Dr. Clemens, there were 1898 cases of chronic poisoning by lead among workmen, admitted into the hospitals of Paris. (Casper's *Viertelj.*, 1853, vol. 2, p. 177.) Out of 1330 cases received during five years (1838–42) in the Parisian hospitals, 655 were among the workers in white lead and painters. Of 341, who were workers in white lead, 55 died. The workers in metals—plumbers who handle metallic lead, are but little subject to the disease. Only 22 cases of this kind occurred in the five years. (*Gaz. Méd.*, Janvier 17, 1847.)

The workmen who are employed to whiten Brussels lace by beating white lead into the fibre, constantly breathe an atmosphere of this poisonous salt, and suffer, according to M. Chevallier, from dryness of the fauces, colic, and other symptoms of chronic poisoning by lead. (*Ann. d'Hyg.*, 1847, vol. 1, p. 111, 1855; vol. 2, p. 317.) It becomes a question whether those women who wear this lace in close contact with the skin, may not suffer from symptoms of lead-poisoning. At any rate, those who prepare the lace suffer and die from the effects of lead-poisoning. A fatal case of this kind is reported in the *Med. Times and Gazette.* (Dec. 19, 1857, p. 636.) Lead was found in the viscera of a woman who had thus fallen a victim to lace-whitening. (*Gazette Médicale*, Dec. 11, 1847, p. 993; and *Ann. d'Hyg.*, 1856, vol. 2, p. 316.) Actors and others who use white lead as a cosmetic, to give a delicate paleness to the countenance, are liable to attacks of lead-colic. (*Med. Times and Gazette*, August, 1852, p. 223.) The makers of glazed cards, in which white lead is largely employed, also suffer from this disease. So easily is the system affected in some cases that colic and paralysis have been known to arise from a person working or sleeping in a recently painted room. (*Lancet*, Oct. 26, 1844.) In a case reported by Dr. Chowne, a man who slept in a newly painted room for a few nights was attacked with paralysis. (*Med. Gaz.*, vol. 39, p. 255.) In these instances, the noxious emanations are received through the lungs. Sir James Alderson mentions several cases of a similar kind, and he calls this form acute paralysis from lead. (*Lancet*, Oct. 30, 1852, p. 391.) I have myself suffered from severe colic by breathing the vapor of fresh paint. It is not improbable that, in these cases, the carbonate of lead is carried off in vapor in combination with that of the essential oil of turpentine.

The late Mr. J. Lizars, of Edinburgh, communicated to me the following case. A military officer, æt. 50, fond of painting in oil-colors, worked for some time in a room eight feet square, which had a large stove in it. He was attacked with wrist-drop (paralysis) in December, 1855, and soon afterwards with paralysis in both legs. It appears that his servant always ground his colors, mixed them, and cleaned his brushes. He had had an attack some years before; but from this, by laying aside oil-painting, he completely recovered. In these instances the emanations of lead must have been received through the lungs.

Doubtless, chemists might be found who would undertake to prove by ingeniously devised experiments, that there could be no lead in the air of the room; and coupling their results with the fact that few artists are known to suffer from such symptoms, would contend that lead was not the cause. The symptoms, however, were of the character peculiar to lead-poisoning, and as they disappeared on the removal of the patient to another atmosphere, there could be no doubt about the cause. These insidious effects of lead should be borne in mind by those who deny that any noxious emanations can escape from arsenical papers in inhabited rooms, merely because the greater number of persons who live in them do not suffer, and because some chemists have affirmed that they could detect no arsenic in a volatile state in the atmosphere of the room.

There are numerous other instances in which lead, or its preparations, by mere contact with the skin, have been known to produce the usual results of lead-poisoning. Some of these have been already pointed out in a former part of this work. (See action through the skin, p. 26.) Dr. Todd mentions the case of a man in King's College Hospital, who suffered from lead-palsy. He had been a potman, and the palsy was attributed to the constant handling and cleaning of pewter pots. (*Med. Gaz.*, vol. 48, p. 1047.) For another case, see *Lancet*, Jan. 21, 1860, p. 60. Dr. Johnson, of King's College Hospital, treated a case of lead-poisoning in which the cause was traced to the handling of vulcanized rubber, impregnated with lead to give it a dark color. The man was a trunk-maker, and used this material in his trade. (*Pharm. Jour.*, 1870, p. 426.) The mere handling of lead or its oxides, is therefore sufficient to produce all the effects of chronic poisoning. I have been informed of a case in which a tea-dealer was seized with symptoms of lead-poisoning, and the cause remained long unsuspected, until he admitted that, in the course of his trade, he had the idle habit of placing pieces of tea-lead frequently in his mouth, and crushing the metal between his teeth. In the *Journal de Chimie* (Juillet, 1858, p. 434), the case of a compositor is related, from which it appears, that local paralysis of the right hand was induced in a week as the result of handling new type. The sharp edges of the type produced abrasions of the thumb, fore and middle fingers—a condition which favored absorption. In five days the wrist became progressively weaker; in a week, the hand dropped so that it could not be raised, and there was loss of power to grasp any article with firmness. There was a faint blue line at the edge of the gums. This appears to have been an instance of purely local action affecting only one hand, and not preceded by colic. It was found that in this case baths of the sulphide of potassium effected a cure. Cosmetics and hair-dyes, containing preparations of lead, may also insidiously give rise to chronic poisoning.

Plumbism.—The *symptoms* of chronic poisoning by lead are well marked. There is first pain, with a sense of sinking, commonly in or about the region of the navel—the seat of the colon. Next to pain there is obstinate constipation, retraction of the abdominal parieties, loss of appetite, thirst, dryness of the mouth and throat, fetid odor of the breath, sallowness of the countenance, and general emaciation. The skin is dry, acquires a sallow or dusky color, and the patient experiences a saccharine, metallic, or astringent taste in the mouth. There is emaciation and a general wasting of the muscles of the body, especially the arms.

A symptom of a peculiar nature was pointed out by the late Dr. Burton (*Med. Gaz.*, vol. 25, p. 687), namely, a *blueness* of the edges of the *gums*, where these join the teeth, which are generally of a brownish color. This coloring of the gums has been so frequently observed, that pathologists regard it as a well-marked, although not a certain, indication of lead-poisoning. A similar blue mark round the edges of the gums has been noticed in other cases of poisoning—as by mercurial preparations, and as a result of the medicinal administration of the salts of silver. On the other hand, in certain cases of chronic poisoning by lead it may be absent—(see a case by Mr. Fletcher, *Med. Times*, Feb. 14, 1846, p. 395)—as where, for example, the individual has ceased to expose himself to emanations of lead. Dr. Thomson, of Stratford-upon-Avon, has observed, with respect to this coloring of the gums, that it was absent in a case of chronic lead-poisoning in which the other symptoms were well marked. In another instance, in which the poison was derived from the same source, there was no paralysis of the hands, but the gums were deeply tinged. He has also seen this blue tinge of the gums in painters who had not suffered from any of the constitutional effects of the poison. (*Med. Times*, Dec. 1848, p. 195.) In some cases in which the blue color has not appeared, the gums have presented a fungous appearance, and have been observed to bleed frequently. (*Med. Times and Gaz.*, Jan. 30, 1858, p. 124.) Hence, while a blue line indicates poisoning by lead, its absence is not to be taken as a proof that this poison is not in the system.

In April, 1846, a woman was admitted into Guy's Hospital, with some obscure symptoms of chronic gastritis, and dull aching pains in the stomach and back. For ten weeks previously, her bowels had only acted under the use of medicine. On the third day after her admission, a distinctly blue line was for the first time noticed on both gums, and there was trembling of the hands with paralysis of the extensors of the wrists. She became insensible, and died a week after her admission. An analysis of the substance of the liver showed that this organ contained a small portion of lead, and, although no evidence could be obtained that deceased had taken the poison, the chemical analysis confirmed the opinion, derived from the symptoms, that this was a case of lead-poisoning. (*Guy's Hosp. Reports*, 1846, p. 471.) Two cases of plumbism from the grinding of white lead, marked by the usual symptoms, including the blue line on the gums, are reported by Dr. Wilks. (*G. H. Rep.*, 1872–3, vol. 18, p. 155.) An engineer, who had worked for eighteen years in his trade, had during this time used a quantity of red and white lead for various purposes. It was, however, only within the last eighteen months of his work that he had suffered from the usual symptoms of lead-poisoning. (*Lancet*, Jan. 21, 1860, p. 60.) It is worthy of note that, although this person had been so many years exposed to the causes of lead-disease, he did not suffer from any symptoms until the latter part of the time. It appears from the observations of Hitzig that workers in horse hair are subject to attacks of chronic lead-poisoning. The hair is dyed black by a compound containing litharge. (*Ann. d'Hyg.*, 1874, vol. 1, p. 446.) It penetrates through the skin. Other sources of lead-poisoning in manufactures have been pointed out by Dr. Du Mesnil, in reference to the polishing and painting of articles of furniture. (*Ann. d'Hyg.*, 1874, vol. 1, p. 355.) Chronic poisoning by lead often kills the patient; after death the large and small intestines have been found much contracted, and their coats thickened. These changes have been especially observed in the colon.

It is not improbable that many cases of supposed cerebral, spinal, or heart disease, are really due to the insidious and unsuspected action of lead upon the system. In December, 1856, a man was admitted into Guy's Hospital under the late Dr. Addison. He was a pale, exsanguined man, who had suffered from head-symptoms, failing power in the limbs, irregular pains in the stomach, and palpitation of the heart. In looking at his tongue, it was observed that there was a blue line on the gums, and on further inquiry, it turned out that the man had been a worker in red lead, and had suffered much from constipation. The arms had been more affected by the loss of power than the legs—one of the peculiar features of lead-palsy. The case was thus recognized and treated as one of lead-poisoning: the blue line on the gums having first led to a suspicion of its real character. (*Med. Times. and Gaz.*, Dec. 27, 1856, p. 643.) In a case which was under Dr. Rees, at Guy's Hospital, in January, 1861, no source of lead could be traced, although the symptoms were those of chronic lead-poisoning, and lead was found in the urine.

The earliest period at which this blue line on the gums first shows itself is not known; it is the result of a slow deposit of absorbed lead, and in an early stage, unless specially looked for, may escape notice. According to some authorities, the period at which the line is first seen, varies with the quantity of lead taken, as well as the rapidity of absorption. Dr. Burton states that the discoloration has been produced in twenty-four hours, by giving four doses of five grains each of the acetate of lead in six hours; and he thinks it might be obvious in four or six hours when large doses are taken. (*Med.-Chir. Trans.*, vol. 23, p. 78; and Harrison on *Lead Poisoning*, p. 59.) When once produced, this appearance is very persistent. Dr. Thomson noticed it in the case of a patient poisoned by drinking water containing lead, about a year previously; and in a severe case of colica pictonum with slight paralysis of the hands, occurring in a house-painter, after repeated attacks, a faint indication of it was recognized four years after the man had ceased to be subjected to the influence of the poison. In this instance, the color had, in great part, disappeared at the end of the first year. (*Med. Times*, December, 1848, p. 195.)

Among the symptoms is a marked effect on the nervous system. There is a dull, numb feeling in the skin, especially noticed in the fingers and forearms, trembling of the arms and legs, unsteadiness in walking or in any muscular exertion, with rheumatic pains in the loins. Symptoms affecting the brain also present themselves. The loss of power in the wrists is manifested chiefly by a paralysis of the extensor muscles, so that the hand drops. The body becomes emaciated, the legs œdematous, and the person dies exhausted.

A common cause of chronic poisoning by lead is the use of *water* which has acquired an impregnation of a salt of lead from being kept in a cistern or conveyed through a pipe of this metal. The symptoms in these cases come on very insidiously, and are not materially different in their course and character from those above detailed. Dr. Thomson has well described them in the paper to which I have referred. They are manifested by pains in the stomach and bowels, constipation, evacuations when passed dark-colored, headache, flushing of the face, pains in all the joints and limbs, but especially the wrists, with great depression of spirits, paralysis of the extensor muscles of the arms and hands, countenance dark and sallow, eyes sunk, tongue flabby, indented, and of a whitish livid color, with a dark blue line along the transparent edge of the gums. It is obvious that symptoms of this nature may be overlooked or referred to some other cause. In 1857, a man was admitted into Guy's Hospital, laboring under some of the symptoms above described. He had also a blue line at the edges of the gums. So far as it could be ascertained, he had been exposed to no cause of poisoning by lead. He lived at Norwood, and on requiring for examination a sample of the water which he had been in the habit of drinking, it was found to be impregnated with lead derived from a leaden cistern. In these cases the cause is not commonly suspected until the symptoms are fully developed; and it is only after the patient has suffered for some time from the effects of the poison, that the symptoms assume the form of lead-colic. (Harrison, *On the Contamination of Water by Lead*, 1852, p. 60.)

A remarkable series of cases presenting this form of chronic poisoning by lead occurred in the members of the ex-royal family of France at Claremont, in 1848. A full account of the symptoms by Dr. De Mussy, will be found in the *Dublin Quarterly Journal*, May, 1849, p. 405; *Med. Gaz.*, vol. 44, p. 260; and Harrison, *On Lead Poison*, p. 122. Out of thirty-eight persons using water impregnated with lead to the extent of about one grain in the imperial gallon, thirteen were attacked with symptoms of chronic poisoning, but presenting various shades of difference. There was a sallow complexion, yellowness of the eyes, wasting of the body, with frequent attacks of colic, nausea, and obstinate constipation. Among the nervous symptoms, there was great restlessness, depression, and, in some, an excessive sensibility of the skin. As confirmatory of the statement above made respecting the blue line on the gums (*ante*, p. 400), this symptom existed only in one-half of the patients, and there were others in whom the blue line had existed, who did not experience any inconvenience from the lead. Lead was not only found in the water in small proportion, but in the urine of some of the patients; showing that it was undergoing elimination by this secretion. It is remarkable that six children of the family, aged from three to seven years, did not show any symptoms. In some of the patients Dr. De Mussy observed, in addition to a blue line on the gums, slate-colored spots on the mucous membrane of the inside of the mouth.

In one case the mucous membrane of the mouth and tongue of a worker in white lead was observed to be entirely of a slate-blue color.

Effects of External Application.—Some remarks have been already made on this subject (*ante*, p. 399). The application of any preparation of lead in powder, ointments, or diffused in water, is sufficient to excite all the symptoms of plumbism. A nursemaid was in the daily habit of bathing a healthy infant in the distilled water obtained from a leaden pipe connected with a steam-boiler. The child grew up almost paralytic and tottering in gait, and the cause was not suspected until the water was chemically examined and found to contain lead.

Most *hair-washes* or *hair-restorers*, are solutions of acetate of lead, in the proportion of four to six grains to the ounce, mixed with a little sulphur, and colored and scented. Powders consisting of a mixture of lime and oxide or a subsalt of lead, are also used for a similar purpose. (See *Ann. d'Hyg.*, 1832, vol. 2, p. 324.) The long-continued use of these preparations may give rise to symptoms, for the origin of which a practitioner might not be able to account. In one instance a dye thus used proved fatal, and lead was found in the liver and in one of the kidneys of deceased. (*Pharm. Jour.*, Nov. 1869, p. 304; and Jan. 1869, p. 440.) Dr. Brück, of Hanover, observed that a violent ophthalmia was induced in a lady who had used for dyeing her hair a substance called *Poudre d'Italie*, which on chemical analysis was found to consist of oxide of lead and lime. (*Med. Gaz.*, Nov. 1842.) In a case which occurred under my observation, a lady had paralysis of the muscles on one side of the neck, as a result of using a mixture of lime and litharge to her hair. When this was discontinued, she quite recovered. Mr. Lacy has pointed out the injury to health which is likely to follow the use of white lead as a cosmetic by actors. The glazed white leather lining of hats is strongly impregnated with carbonate of lead, which may penetrate the body through the perspiring skin. Other facts connected with this form of lead-poisoning will be found in the *Medical Times and Gazette*, August, 1852, p. 223; *Ann. d'Hyg.*, 1859, vol. 1, pp. 95, 296; also *Ann d'Hyg.*, 1861, vol. 1, pp. 342, 389; and 1870, vol. 1, p. 72. Among the cases mentioned by Orfila, is one of a woman who was in the habit of applying for a long period to her face and neck a cosmetic containing a preparation of lead. After six months there were the usual symptoms of chronic poisoning. This woman ultimately became blind and paralytic, and soon afterwards died. In another instance, the symptoms had become so firmly established before the cause was suspected, that no treatment sufficed to relieve them. (*Toxicologie*, vol. 1, p. 680.) Notwithstanding these facts, M. Tanquerel does not consider that serious symptoms can be produced by preparations of lead coming in contact with the unbroken skin. If the skin be abraded, then absorption may take place rapidly. A case is reported by Taufflieb, in which the frequent application of lead plaster to an ulcer of the leg was followed, in less than three months, by all the symptoms of chronic poisoning. (*Galtier*, vol. 1, p. 698.) The use of a simple lead-wash in cutaneous disorders has not been attended with any injurious effects; but the application of white lead and linseed oil to an abraded surface, produced in one instance an attack of lead-colic. (*Beck's Med. Jur.*, vol. 2, p. 650.)

Appearances after Death.—The appearances observed in cases of acute poisoning by lead are very characteristic. The mucous membrane of the stomach and intestines is covered with a thick white or whitish-yellow layers of mucus mixed with the salt of lead, and beneath this, the membrane is reddened or ecchymosed. In the fatal cases of poisoning by subacetate of lead (*ante*, p. 394), the following appearances were found. The mucous membrane of the stomach was of a gray color, but otherwise perfectly healthy. The intestines were found much contracted, in one instance more so than in the other. A case is reported by Orfila, in which an inspection was made of the body of a man who had been killed by taking a quantity of Goulard's extract. He died within forty-eight hours, and there was well-marked inflammation of the alimentary canal from the œsophagus downwards. The inner coat of the stomach was completely softened, and the effused mucus was found to contain the poison. (*Toxicologie*, vol. 1, p. 671.)

In a case related by Dr. Kerchoffs, the mucous membrane of the stomach was found abraded in several places, especially near the pylorus; and most of the abdominal viscera were in a state of high inflammation. A trial for murder by this substance took place at the Central Criminal Court in November, 1844 (*Reg.* v. *Edwards*). In this case the stomach and intestines are stated to have been found inflamed, and there were dark spots on the former. In animals, according to Dr. Mitscherlich, when the dose is large, the mucous coat of the stomach is attacked and corroded; this change appears to be purely chemical, and takes place in all the organs of the body with which the salt of lead comes in contact. If given in a small dose it is decomposed by the gastric secretions, and exerts no corrosive power on the mucous membrane. When the acetate of lead was given in a state of albuminate dissolved by acetic acid, death took place with great rapidity; and, on inspection, the stomach was not found corroded. The corrosive action is a property of the neutral salt, and is not manifested when the dose is small, or when the poison is combined with an excess of acid. In the case of poisoning by the carbonate of lead, which proved fatal in ninety hours (*ante*, p. 395), the mucous membrane of the stomach was much inflamed and of a dark red color throughout.

In the *chronic* form of poisoning, the appearances are less distinct. The blue line on the gums may or may not exist in the dead body, according to circumstances. In the case of the woman elsewhere related (*ante*, p. 400), there was thickening as well as enlargement of the coats of the stomach especially of the mucous membrane. The large intestines were irregularly contracted and distended. There were spots of congestion upon the mucous coat, and the salivary glands were enlarged. In Dr. Letheby's case (*ante*, p. 397), the skin was of a dingy yellow, and the gums were of a deep blue color. The lungs were slightly congested, and there was an effusion of serum in the pleuræ. The blood was black and liquid. The stomach and intestines were pale and nearly empty; the former contained half an ounce of a thick brownish fluid, in which lead was detected; the latter were contracted in some places and distended in others, and they presented several points of intussusception. The large intestines were in a similar condition. The bronchial and mesenteric glands were enlarged. The bladder and ureters were full of urine; the rest of the viscera healthy.

Böcker describes the mucous membrane of the stomach and intestines as congested and softened, presenting often a yellow, gray, brown, or black appearance. The coats of the intestines are thickened, and the canal is irregularly contracted. The lungs are congested; the muscles are pale, wasted, and converted into a fibrous tissue. (*Vergiftungen*, 1857, p. 49.) For further information on this subject, I must refer the reader to the *Traité des Maladies de Plomb*, par Tanquerel des Planches, 1839; *Traité Pratique de la Colique de Plomb*, par J. L. Brachet, 1850; and a translation of the work of Tanquerel des Planches, by Dr. Dana, U. S., 1848.

Fatal Dose—Period of Death.—Nothing is accurately known concerning the fatal dose of sugar of lead, or of carbonate of lead. The facts already detailed show that either substance may be taken in a comparatively large quantity, without producing serious effects. Thirty and forty grains of the acetate have been given daily, in divided doses, without injury. The following additional cases, in some of which recovery took place under very disadvantageous circumstances, prove that sugar of lead is not an active poison. The late Dr. Iliff met with an instance in which *an ounce* was swallowed in solution. The symptoms were pains in the abdomen, resembling colic, with vomiting, muscular rigidity, and numbness. It was three hours before any remedies were used, and five hours before the stomach-pump was employed. The person recovered. In a second case an ounce was swallowed; sulphate of magnesia was freely given; the stomach-pump was used, and the patient recovered. In October, 1835, a girl, æt. 19, dissolved an ounce of acetate of lead in a cupful of water, and swallowed it. In a quarter of an hour violent vomiting came on, and she was taken to the North London Hospital. Sulphate of magnesia and diluted sulphuric acid were given to her. There was slight pain in the abdomen, weight in the head, dimness of sight, with pains shooting through the eyeballs. The abdomen was tender on pressure for several days; but in five days the patient was discharged cured. The fourth case occurred in Paris, in 1840. A girl swallowed *an ounce* of sugar of lead; the usual symptoms followed, and sulphate of soda was administered. She recovered. In a case reported by Dr. Evans, a woman recovered after having swallowed half an ounce of the acetate by mistake, under the free use of aromatic sulphuric acid. (*Amer. Jour. Med. Sci.*, Feb. 1847, p. 259.) Mr. Marshall mentions a case of recovery where two fluid ounces of Goulard's extract had been taken by mistake. (*On Arsenic*, p. 106.)

The dose of this poison required to destroy life was a subject of inquiry in *Reg.* v. *Hume* (Chelmsford Summer Assizes, 1847). The prisoner was charged with an attempt to murder her husband, by endeavoring to administer to him "a large quantity of a certain *deadly poison*, called sugar of lead!" According to the evidence, she made two boluses with flour and water, and the quantity of sugar of lead contained in them was equal to *twenty-six grains and a half*. This was pronounced to be sufficient to destroy life, although the grounds for this medical opinion did not appear. So far as I have been able to ascertain, there is not a single instance recorded in which even sixty grains have destroyed life. Van Swieten gave it to the amount of one drachm daily, for ten days, before it caused any material symptoms. (See *Christison, op. cit.*, 555.) In another case, violent symptoms were produced by this dose; but the individual easily recovered from the effects. The observations and experiments of Orfila also prove that the vulgar belief of sugar of lead being an active poison is erroneous. The fatal cases are so few in number, that it is impossible to fix, with any precision, the period within which this poison may destroy life.

Treatment.—This consists, in acute poisoning, in the free administration of solutions of the alkaline sulphates, either of soda or magnesia. The carbonates should be avoided, as the carbonate of lead is poisonous; while the sulphate is either inert, or possesses but very little activity. Purified animal charcoal has been recommended as an antidote, in consequence of the property which it possesses, to a certain extent, of separating oxide of lead from its saline combinations; but there is no record of its efficacy or utility.

An emetic of sulphate of zinc should be given if vomiting does not already exist; and castor oil may be given to promote free evacuation from the bowels. The stomach-pump may be occasionally employed with benefit. It is well known that albumen precipitates the oxide of lead when added in large quantity; and Mitscherlich has found that casein, the albuminous principle of milk, is an effectual precipitant of the oxide of lead. Therefore it would be advisable to administer, in cases of poisoning by the soluble salts of lead, milk or albumen in large quantity. The compounds thus formed, as in the case of corrosive sublimate, may not be absolutely inert; but they are far less active than the acetate itself, and tend to prevent the action of the poison as a corrosive on the stomach. Six cases have been mentioned in which the patients recovered partly through treatment, after having swallowed one ounce of the acetate of lead. M. Bouchardat strongly advises the employment of the hydrated persulphide of iron as a chemical antidote. This compound may be made by adding a persalt of iron to an alkaline persulphide, the latter being in excess. It should be mixed with syrup, and preserved closely bottled, to prevent chemical change. It is said to be inert, and may be given in large quantity. (*Annuaire de Thérapeutique*, 1847, p. 297.) The urine should in all cases be chemically examined for the purpose of tracing the disappearance of the poison from the body. As a chemical antidote in poisoning with carbonate of lead, a mixture of vinegar and sulphate of magnesia may be employed.

In the treatment of *chronic* poisoning, the principal object is to remove the poison from the stomach. In a case of poisoning with water impregnated with lead, another source of supply should be immediately provided. The use of dilute sulphuric acid internally, and the most scrupulous attention to cleanliness of the skin by frequent ablutions, have been found the best means of preventing and treating some forms of chronic poisoning. When the poison in once absorbed, nothing can be done beyond trusting to its elimination through the urine and other secretions. The use of iodide of potassium has been strongly recommended, on the ground of its dissolving the lead, and carrying it off by the kidneys; but there is no satisfactory evidence that it has in any case accelerated a cure. (*Lancet*, Dec. 3, 1853, p. 522.) There is probably no metal which is retained so long in the body as lead, when it has been once deposited in the tissues. (See *post*, p. 410.) The use of sulphuretted waters, or alkaline sulphuretted baths, is, under these circumstances, just as inefficacious as the use of diluted sulphuric acid. (See *Orfila*, *op. cit.*, vol. 1, p. 686; *Galtier*, *op. cit.*, vol. 1, p. 676.) Dr. De Mussy employed sulphur baths in the treatment of the royal family of France, but all that he effected was a blackening of the nails and skin by the production of the sulphide of lead! MM. Legroux and Girard have, however, employed these sulphur-baths, and, as they believe, with benefit. In some lead-works at Marseilles, in 1853, fifty-two out of 260 workpeople were affected with chronic poisoning within a month. Croton oil was given frequently, and sulphur-baths were employed towards the termination of the case. Two or three weeks were required for internal and external depuration in these cases of lead-poisoning. (*Med. Times and Gaz.*, Jan. 30, 1858, p. 125.)

Other modes of treatment will be found described by the writers to whose works on chronic poisoning I have already referred.

CHAPTER XLIII.

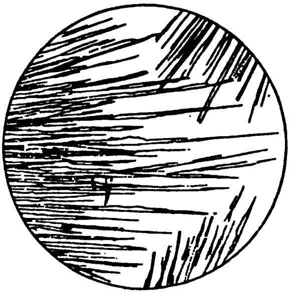
CHEMICAL ANALYSIS OF THE SALTS OF LEAD—ACETATE AS A SOLID AND IN SOLUTION—LEAD IN ORGANIC MIXTURES—IN THE TISSUES—IN ARTICLES OF FOOD—ACCIDENTS FROM SPURIOUS TIN-FOIL.

CHEMICAL ANALYSIS.

Acetate of Lead as a Solid.—1. If a portion of the powder is heated in a small reduction-tube, it melts, then becomes solid; again melts, acquiring a dark color, and gives off vapors of acetic acid; a black mass is left in the tube, consisting of carbon and reduced metallic lead. There is no metallic sublimate formed. 2. It is very soluble in water, even when cold; river or spring water is turned milky by it, chiefly from the presence of carbonic acid and sulphates. Goulard water is generally opaque for this reason. 3. A small portion of the powder placed in a saucer, containing a solution of iodide of potassium, acquires a fine yellow color. 4. When treated with caustic potash, it remains white. 5. By sulphide of ammonium, or sulphuretted hydrogen water, it is turned black, in which respect it resembles the white salts of some other metals. 6. When the powder is boiled in a tube with diluted sulphuric acid, acetic acid, known by its odor and volatility, escapes.

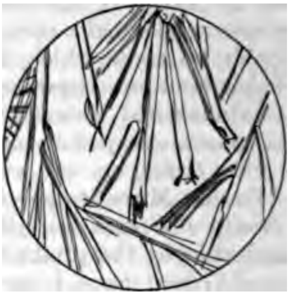
All these properties, taken together, prove that the salt is *acetate of lead.*

FIG. 35.



Crystals of acetate of lead, magnified 30 diameters.

FIG. 36.



Crystals of acetate of lead magnified 80 diameters.

Acetate of Lead in Solution.—If acetate of lead is presented in a state of solution, or if the solid salt is dissolved in water for the purpose of making a further examination, we should note the following points: 1. A small quantity, slowly evaporated on a glass-slide, will give white and opaque prismatic crystals (Figs. 35 and 36), which are turned yellow by iodide of potassium, and black by sulphide of ammonium. The solution is said to be neutral. The common acetate has both an acid and an alkaline reaction, *i. e.*, it reddens litmus-paper and renders turmeric brown, a circumstance which might create some embarrassment in an analysis. 2. *Diluted sulphuric acid* produces a white precipitate, soluble in hydrochloric acid and in a large excess of caustic potash. 3. It is precipitated of a bright-yellow color by the *iodide of potassium;* the yellow iodide of lead is soluble in caustic potash as well as in concentrated hydrochloric acid, forming colorless solutions. 4. *Sulphide of ammonium*, or a current of sulphuretted hydrogen gas, produces in acid and very diluted solutions, a deep brown-black precipitate. This effect is observed when less than the 100,000th part of the salt is dissolved. The potash solutions of the sulphate (2) and of the iodide (3) are precipitated black by these tests. 5. Place a few drops of the solution in a clean platinum capsule; acidulate it with acetic acid, then apply, through the solution, to the surface of the platinum, a thin polished slip of zinc—bright crystals of metallic lead are instantly deposited on the zinc. 6. Zinc alone, placed in an acid solution, slowly displaces the lead. The metal may be thus obtained in a dark blue spongy mass. Although this is not a delicate mode of testing, yet by it the metal may be detected and separated from the liquid.

Among these tests, there is none so efficacious or certain as the sulphuretted hydrogen gas. A current of this, when properly applied, will reveal, by the production of a brown tinge, a quarter of a grain of a salt of lead in a gallon of water, *i. e.*, about the 300,000th part. In operating with the other tests on small quantities of lead-salts, it is always advisable to concentrate the liquid to the smallest possible bulk, and even in some cases to apply the tests to the dry residue.

Lead in Organic Mixtures.—The acetate of lead is precipitated by many organic principles, especially by casein, albumen, and tannic acid. Thus we may have to analyze either an organic liquid containing lead, or a solid precipitate consisting of mucus or mucous membrane, intimately united to oxide of lead. If the liquid should be deeply colored, and mixed with much organic matter, such as blood or mucus, it may be submitted to dialysis in the manner already described for other metallic poisons (p. 154). In this way if any lead is dissolved, a solution may be obtained so clear as to admit of the direct application of the above tests. As a trial test some portion of the dialyzed liquid, acidulated with nitric acid and placed in a platinum capsule, may be treated with zinc. When the zinc and platinum come in contact, metallic lead will be separated.

As all organic liquids, such as wine, vinegar, beer or cider, containing a salt of lead in solution, acquire a dark-brown color from sulphuretted hydrogen, this gas may be generally employed as a trial test. For this purpose a small quantity of the liquid, diluted if necessary, may be used. If thus detected in a portion, the whole of the lead may be precipitated from the solution as black sulphide of lead. The precipitate should be collected on a filter, washed and dried, then boiled for a quarter of an hour in a mixture of one part of nitric acid diluted with four parts of water. This has the effect of transforming it, at least in part, into nitrate of lead soluble in water. This liquid, when filtered, may be evaporated to dryness, and the residue dissolved in water, or it may be at once cautiously neutralized by potash (free from lead) or by ammonia, and the tests applied. If the quantity is too small for the application of all the tests, we may first add diluted sulphuric acid; should a white precipitate be formed, soluble in potash (free from oxide of lead), and this alkaline solution be again turned black by sulphide of ammonium, this is sufficient evidence of the presence of lead. Should there be no lead dissolved, we must decompose the solid and insoluble matters in nitric acid, slightly diluted, at a boiling temperature, filter, and test the filtered liquid, previously neutralized.

In the Tissues or the Urine.—The organic matter, such as a part of the liver or other organs, should be dried and incinerated in a porcelain vessel. The ash should be heated with a small quantity of strong nitric acid, and then evaporated to dryness. The nitrate of lead thus formed, may be dissolved out of the residue by water and filtered. A portion of this liquid evaporated on a slide will yield crystals of nitrate of lead which may be identified—1, by covering them with a solution of iodide of potassium. If lead, they acquire a brilliant yellow color. 2. A solution of sulphide of ammonium renders them black. The remainder of the liquid, after filtration, may be treated with a current of sulphuretted hydrogen gas. A brown color or a brown precipitate, not readily dissolved by nitric acid, indicates the presence of lead. The metal itself may be obtained, if necessary, by plunging zinc into a portion of the acidulated liquid.

Lead may thus be detected in the tissues, in all solid articles of food, and in the dry residues obtained from the vomited matters, the contents and coats of the stomach, the urine, and other liquids.

The subacetate of lead, Goulard's water, and the other soluble salts of the metal, may be analyzed by a similar process. The acids of the salts may be discovered by the tests elsewhere described. The nitrate of lead, when heated, yields red vapors of nitrous acid; the chloride fuses and forms a fixed greenish-yellow mass; the carbonate yields an orange-colored residue of oxide of lead. Litharge may be examined by dissolving it in diluted nitric acid, and minium or red lead by digesting a portion of it in strong nitric acid.

Sometimes the carbonate or sulphate of lead (the latter as a result of antidotal treatment) are found in the form of a white powder in the stomach. They may be collected by decantation, and examined in the manner above pointed out.

Quantitative Analysis.—This may be most conveniently effected with respect to any of the soluble salts of lead, by passing into the solution a current of sulphuretted hydrogen gas, until the filtered liquid gives no longer any indication of the presence of lead. The precipitate should be well washed, dried, and weighed. Every 100 parts of sulphide of lead are equal to 158.3 of crystallized acetate; 138.3 of crystallized nitrate; 116.6 of chloride, and 111.6 of carbonate of lead. In some cases it may be convenient to precipitate the lead by sulphuric acid, and to calculate the quantity from the sulphate washed and dried. One hundred parts of this salt are equal to 26.4 of sulphuric acid, and 73.6 of oxide of lead. It has been alleged that lead is a *normal* constituent of the body. In reference to this allegation, it may be remarked that the lead hitherto found in the tissues is not normal, but of an abnormal kind; and had the history of such cases been properly brought out, its introduction *ab extra* would have been demonstrated.

Absorption.—Both in acute and in chronic cases of poisoning, the metal lead, in some form, is found, more or less, in all the soft organs of the body. The blue line on the gums, where they join the teeth, affords an instance of its deposition in these parts—the color being probably due to the conversion of the deposited lead into the state of sulphide. Lead was found by Tiedemann, in the blood of poisoned animals, and Prof. Cozzi found it in the blood of a person laboring under lead-colic. Flandin did not succeed in detecting it in this liquid. The urine appears to be the great channel of elimination. Orfila found lead in the urine, in the case of a woman who had swallowed an ounce of the acetate (*Op. cit.*, vol. 1, p. 684), but Dr. Mitscherlich could not find it in the blood and urine of animals which he poisoned. In the case of a cow poisoned by lead-paint, I found traces of it in the milk. Dr. Letheby states that, in a case of chronic poisoning, he detected lead in the brain, muscles, liver, and intestines, as well as in blood and serum found effused in the ventricles of the brain; but none was discovered in the bile or urine. Dr. Inman detected it in one case in the cerebellum. (*Med. Gaz.*, vol. 38, p. 389; see also, for its detection in the brain, *Ed. Monthly Journal*, July, 1851, p. 65.) In a case elsewhere related, lead was discovered in the liver of a woman, and the symptoms from which she had suffered thus received an explanation. The metal has been detected in the tissues a fortnight after the taking of the lead poison had ceased. M. L. Orfila states that he found it in the tissues so long as eight months after the withdrawal of the poison; and the facts connected with the slow disappearance of the blue line from the gums in poisoning with this metal, render it probable that it may be detected after the lapse of one or two years.

Although there are no facts to show that lead is a natural constituent of the body, yet the metal may be found in the tissues, in cases in which there can be no suspicion of criminal administration. Next to copper, no metal is so frequently met with in various articles of food as this (see *post*, p. 412); and as it is more slowly eliminated than other metals, it may accumulate in the tissues, and be occasionally discovered after death by chemical analysis. It is impossible, however, to raise a charge of poisoning on such a discovery. In November, 1843, a trial took place at the Assizes of the Puy de Dôme, in France, involving this question. The deceased died under suspicious circumstances; on an inspection of the body there was nothing to indicate the action of an irritant poison, but the stomach was ulcerated and in an otherwise diseased condition. No salt of lead was found in the contents, but *traces* of the metal were discovered on incinerating the viscera. The question then arose whether the metal thus found was a natural constituent of the body, or the result of a portion which had been swallowed, and had acted as a poison. The medical opinions were conflicting. Orfila thought it was very probable, if not certain, that the deceased had died from the effects of lead (*Annales d'Hygiène*, Janvier, 1844); but the traces of lead were probably due to accidental causes—they may have been taken in water, wine, snuff, or some other article of daily use.

Lead has been so frequently detected in the soft organs and secretions, that the presence of it in the tissues of the body may now be looked for with some certainty, when in a case of poisoning it cannot be discovered either in the matter vomited or in the contents of the stomach after death. I believe that the liver, from its size and from the large quantity of blood it contains, is the organ best adapted for analysis. Drs. Wilson and Macadam infer from their experiments that the largest proportion of deposited lead will be found in the spleen, but this was in reference to a solitary case. They assign the following as the order of maximum deposit in the tissues,—the spleen, liver, lungs, kidney, heart, and coats of the intestines. Here, as in the incineration of any of the soft parts of the body, the analyst is liable to be embarrassed by the presence of oxide of iron in the ash. This oxide may give a color with sulphuretted hydrogen gas, which, if this test alone were employed, might easily lead to error. The sulphides of lead and iron are, however, very differently affected by nitric acid; and on making the liquid rather strongly acid with this menstruum, we are quite sure that no sulphuret of iron will be formed. In an acid mixture of these two metals brown sulphide of lead only is precipitated. In searching for the metal in the tissues, it is proper to remember that lead may be introduced accidentally into the ash by a crucible, or by other means which will easily suggest themselves; and as the tests for lead are of exceeding delicacy, it is the more necessary to use great caution in the steps of an analysis. (See *Edin. Monthly Journ. Med. Sci.*, 1852, vol. 14, pp. 386, 389.)

In reference to the presence of lead in organic solids—bread, cheese, snuff, etc.,—the only plan of detecting the metal is to burn the organic substance, or to decompose it by heat, and to digest the carbonaceous ash in nitric acid, slightly diluted. The acid liquid should be filtered, and then tested by the appropriate tests (p. 409). Unless the incineration is complete, errors may arise. (*Ann. d'Hyg.*, 1874, vol. 1, p. 161.)

Lead as an Accidental Ingredient in Articles of Food in Daily Use.—Liquids used for culinary or dietetic purposes, especially if they contain a free acid, are liable to become impregnated with oxide of lead, derived from the *glaze* of the vessel in which they are kept, and thus to form poisonous salts. If vinegar is used, acetate of lead may result.

Litharge-glaze is easily dissolved by acid, alkaline, or fatty substances. The eating of dripping, or the fat of meat, baked in a newly glazed vessel, has thus been known to give rise to a slight attack of colic, while the symptoms were referred to some substance mixed with the food. A case, in which the whole of the members of a family were thus poisoned, will be found reported in the *Lancet* (July 4, 1846, p. 27). Another instance of a similar kind is reported in the *Medical Gazette* (vol. 47, p. 659; also *Lancet*, 1860, vol. 1, p. 962). All newly glazed vessels yield a larger or smaller proportion of lead on boiling in them pure acetic acid, or a solution of potash free from lead. In this manner the poisonous nature of the glaze may be tested, the oxide of lead being dissolved by the acid or the alkali. I have found common acetic acid itself to contain, as impurity, two per cent. of acetate of lead. I have also found lead in crystallized citric and tartaric acid, and in salts crystallized in leaden pans.

Lead may be an accidental ingredient in distilled water, in the waters of the essential oils, as well as in certain medicines. Some of these acquire an impregnation of it in the process of manufacture; *e. g.*, carbonate of ammonia, which is sublimed into leaden vessels; carbonate of soda, borax, and other salts, when crystallized in leaden pans. M. Chevallier thus found tartaric acid contaminated with lead to the extent of 1.2 to 1.5 per cent. He believes that this arises from the employment of lead to sink the strings in the crystallizing solutions. (*Journal de Chimie*, Juin, 1858, p. 354.) Solutions of alkaline salts kept in flint-glass bottles generally contain lead. The alkalies, potash and soda, their carbonates and bicarbonates, the alkaline silicates, phosphate of soda, and some others, are thus rendered impure and unfit for chemical use; but the quantity of lead present is not sufficient to produce symptoms of poisoning.

I am indebted to Mr. Procter, of York, for the particulars of a case of some novelty, in reference to the contamination of food with lead. In July, 1852, four men partook of rhubarb-pie and *milk* for supper. Shortly afterwards they were all seized with violent vomiting and intense colic. A portion of the vomited matters and food was examined by Mr. Procter, and lead was detected in them. The only source to which the lead could be traced was the litharge glaze of the pans in which the milk was kept.

Leaden pipes are largely used by publicans for the supply of *beer*. It is possible, therefore, if the beer is acid, and it is allowed to remain some time in the pipe, that it may acquire an impregnation of lead, and the first portions drawn may give rise to colic and other unpleasant symptoms, creating a suspicion of criminal poisoning. *Cider*, which is a highly acid liquid, is apt to become poisoned with the salts of lead owing to the use of leaden vessels or pipes in its manufacture. An instance of the fatal effects of cider so poisoned, is reported to have occurred in Worcestershire in Jan. 1864, and another fatal case occurred in Herefordshire in 1867. Eight men were seized with symptoms of lead-poisoning, and one died. The late Mr. Herapath found one grain of lead-salt in a gallon of the cider. The leaden pipe was found corroded by the acid of the cider. When liquids of this kind are impregnated with oxide of lead, the fact is immediately made known by their being turned of a brown color by sulphuretted hydrogen. (See a paper by Dr. Waldman, of Erfurt, Horn's *Vierteljahrsschrift*, 1770, vol. 1, p. 268.) It has been generally supposed that the only poisonous compound produced by cider is the insoluble malate; and it appears from an accident which occurred in France, whereby six persons were seized with symptoms of lead-poisoning from drinking cider, that Chevallier and Ollivier discovered that the salt which caused the symptoms was the malate of lead. A large quantity of acid may probably dissolve this and other vegetable salts which are reputed to be insoluble; or, like the carbonate of lead in water, the insoluble salts may be diffused through the liquid, and thus taken in an extreme state of division. In some instances the carbonate of lead itself may be formed and act as the poison. A case of this kind has been already related. (*Ante*, p. 396.) An instance of chronic poisoning occurred in an American family, by reason of the members of it drinking cider which had been poured into vessels newly painted with white lead and linseed oil. No effect was observed for a fortnight. The chief peculiarity in the symptoms, was a soreness in the soles of the feet, succeeded by slight nausea and a vesicular eruption. (*Amer. Jour. Med. Sci.*, July, 1843; and *Trans. Prov. Assoc.*, vol. 1, p. 119.) In another set of cases colic and constipation followed the use of cider which had acquired an impregnation of lead from its having been passed through a leaden funnel. Lead was detected in the cider in the proportion of a 4000th part, or about seventeen grains in the gallon. The urine of one of these persons was found to contain lead. (*L'Union Médicale*, Feb. 17, 1857, and *British and Foreign Medical Review*, vol. 19, 1857, p. 499.) In a case heard at Glasgow, Dec. 1874, it was proved that some lemonade which was sold as genuine contained lead in the proportion of one-sixth of a grain to the gallon. (*Pharm. Jour.*, Dec. 25, 1874, p. 515.) This was traced to the use of leaden pipes and receivers by the maker. He was heavily fined under the Adulteration Act. When acid liquids of this kind are impregnated with oxide of lead, the fact is immediately known by their being turned more or less of a brown color by sulphide of ammonium.

Litharge was formerly much used to remove the acidity of sour *wine*, and convey a sweet taste. Acetate of lead, or some other vegetable salt of the metal, is in these cases formed; and the use of such wine may be productive of alarming symptoms. Many years since a fatal epidemic colic prevailed in Paris owing to this cause; the adulteration was discovered by Fourcroy, and it was immediately suppressed. Such wine is known by its being blackened by sulphide of ammonium.

Lead-shot are much employed for the purpose of cleaning wine-bottles, and pellets are frequently left in the bottles. A question has arisen, whether wine introduced into them is liable to acquire a poisonous impregnation from lead. A case related at page 396 furnishes an answer. I have found, when the shot are in much larger proportion than could ever be left by accident in a wine-bottle, that good wine, whether port or sherry, is slowly impregnated with lead. After two or three months a white sediment had formed, but no lead was dissolved; after thirteen months the port wine retained its color, and scarcely any portion of lead was dissolved in it; the sherry had become darker in color, and the presence of lead was very evident in it. The undissolved salt of lead in the sediment will, however, if diffused through the wine and swallowed, produce all the effects of chronic poisoning. Very acid wines (from acetic acid), such as those made from the currant or gooseberry, may, however, under these circumstances, be rapidly impregnated with the metal, in a quantity sufficient to produce colic or other serious symptoms. *New rum*, as it is made in the West Indies, often contains lead derived from the leaden worm of the still, and lead-colic frequently attacks those who drink it. *Old rum*, on the other hand, is by no means unwholesome, and is therefore in great demand. Dr. Traill gives the following explanation of this difference in properties. He found that the rum which was received in glass bottles from the still, was always impregnated with lead from the pipes; but when kept in oak casks, the tannic acid of the oak is slowly dissolved by the spirit, and precipitates the lead in an insoluble form, as tannate of lead, the spirit thereby becoming perfectly wholesome. He has suggested that a little decoction of oak bark, added to new rum, would render it equally innoxious. (*Outlines*, 112.) Mr. Scanlan has called attention to the fact that oxide of lead is sometimes present in *distilled water*, when leaden pipes have been used for the purpose of condensing the vapor. It appears, however, to be rapidly converted to carbonate, and thus rendered insoluble. (*Pharm. Jour.* Aug. 1844, p. 69; also Dec. 1845, p. 279.)

Pork is frequently salted in leaden vessels, and is allowed to remain in such vessels soaking in the brine. The effect of this is to impregnate the pork with a portion of chloride of lead: its color and taste have been observed to be affected under these circumstances.

Salt Butter.—Mr. Bergeron reported the following cases to the French Academy, August 1874. Twenty-six persons were suddenly seized with symptoms of lead-poisoning, and two of them died. The cause of this was traced to the brine with which the butter consumed by these persons was preserved. The brine contained sea-salt almost to saturation, sugar, nitre, acetate of soda, and chloride of lead. According to some authorities, this is one of the most poisonous compounds of the metal. A notable proportion of lead was found in the liver and intestines of those who died, and also in the brain. (*Brit. Med. Jour.*, Aug. 1874, p. 258.)

Flour.—In 1857, several families at La Tremblaie, in France, suffered from symptoms of lead-poisoning. On analysis, a salt of lead was found in large quantity in the flour and in the bread. An inquiry into the facts led to the discovery that part of the grinding machinery of the mill had been stopped with lead-cement, and this was covered with plaster. The plaster had given way, and the salt of lead which fell out was ground and mixed with the flour. (*Journal de Chimie*, 1857, p. 278.) A similar accident is reported by the American editor of this work. During the year 1866 whole families in one of the counties in the State of New York were poisoned by the use of flour manufactured at a mill the owner of which had been in the habit of filling up the cavities in the millstones with lead. In a set of cases elsewhere referred to (*ante*, p. 394) a salt of lead was added to the flour by mistake.

Sugar.—In 1850, an attempt was made in this country to work a patented process for the refining of sugar by the use of subacetate of lead, the surplus lead being afterwards precipitated in the syrup by a current of sulphurous acid. The late Dr. Pereira, Dr. Carpenter and myself were required by the Government to report on this process in reference to its probable effect on public health. We found that the lead was not entirely removed from the refined white sugar, but that a variable quantity (from two-tenths to four-tenths of a grain in four pounds) remained in it under the form of sulphite of lead; and there was good reason to believe that a still larger proportion was carried into the treacle. Our report was decidedly adverse to the project. It has been found that sugar, as it is ordinarily manufactured, is sometimes a medium of conveying lead-poison into the system, and giving rise to attacks of colic in those who partake of it. Dr. Jackson has reported an instance of this kind, in which several persons lost their lives, and many others were attacked with paralysis and colic, who had partaken of sugar which had probably been kept in leaden reservoirs. Lead was discovered in the sugar in large quantity. (*Med. Gaz.*, vol. 17, p. 1036; see also *Beck's Med. Jur.*, vol. 2, p. 646.) Sugar, refined by the ordinary process, may also contain traces of lead. The metal cones into which the syrup is poured are painted with white lead; and this requires occasional renewal—a proof that the loaf of sugar must be more or less contaminated by contact with the lead—and a portion being thus mechanically taken up. This is a noxious practice, and ought to be prohibited.

Snuff—Tobacco.—Some varieties of snuff are adulterated with lead to a degree to cause symptoms of chronic poisoning. The compounds of lead used for this purpose are the red oxide (minium) and the chromate of lead; the object of the adulterator being to improve the color and the salable value of the snuff, at the expense of the health, and it may be of the life, of his customer. Two instances of chronic poisoning by lead have come under my notice, as a result of the presence of oxide of lead in snuff. One sample contained the oxide in large proportion. This noxious adulteration has frequently given rise to paralysis and other forms of lead disease. (*Med. Gaz.*, vol. 32, p. 138; also *Annales d'Hygiène*, 1831, vol. 2, p. 197, and *Lancet*, Jan. 21, 1860, p. 60.) It is readily detected by incinerating a small quantity of the snuff in a porcelain capsule and digesting the ash in warm nitric acid. This may be afterwards diluted with water and filtered for the application of the tests for lead. (See p. 409.)

Out of forty-three samples of popular kinds of snuff examined by Dr. Hassall, chromate of lead was detected in nine and oxide of lead in three. The chromate varied in quantity from one to four and a half per cent., and the oxide (red lead) reached as much as three per cent. (*Food and its Adulterations*, p. 591.) From this statement it will not be surprising that snuff should occasionally cause symptoms of lead-colic, or even death. (*Med. Gaz.*, vol. 32, p. 138; also *Ann. d'Hyg.*, 1831, vol. 2, p. 197.) In a case in which I was consulted a few years since, I have reason to believe that snuff, adulterated with lead, led to a series of constitutional symptoms which ultimately destroyed the life of a gentleman. This subject has lately been investigated by Dr. Meyer, of Berlin, and the results are most unsatisfactory for the takers of snuff. 1. A man, æt. 38, was seized, without any apparent cause, with paralysis of the extensor muscles of the three middle fingers of each hand. In two months there was a considerable projection of the wrists. It was then discovered that the snuff which he had been in the habit of taking contained a large proportion of lead. The use of this snuff was discontinued, and the paralysis disappeared under treatment. 2. A man, æt. 43, used snuff from the same factory. He suffered from disturbed digestion and colic. In February, 1855, he was attacked with paralysis, involving first the fingers and slowly extending to the muscles of the forearm and shoulders. There was loss of sensation and motion in the extensor muscles, and a yellowness of the skin. This man recovered in a year. 3. A similar case. There were colics, with paralysis of the arms and hands in 1852, and of the legs in 1854. There was also wasting of the extensor muscles. A discontinuance of the snuff led to the disappearance of the colics and improved the condition of the patient. 4. A physician, æt. 45, in the habit of taking snuff. There was complete paralysis of the upper limbs in 1851, and of the lower limbs in 1854. The cause was not suspected; but when discovered and removed there was a rapid cure. 5. A man, æt. 50. There was paralysis of the fingers following the use of snuff, which ceased on the removal of the cause. (*Journal de Chimie*, Juillet, 1858, p. 394.) Dr. Meyer regards the following as the most prominent symptoms: Paralysis, affecting chiefly the extensor muscles of the arms; yellowness of the skin; prominence of the metacarpal bones; colics and weakness, with wasting of the extensor muscles of the hands. The great danger in these cases is that the real cause of the symptoms may escape notice until they are too far advanced for cure.

Apart from wilful adulteration, *snuff* and *tobacco* are liable to acquire an impregnation of lead from being kept in vessels or wrappers made of lead, or of a spurious alloy of lead, called "patent tin-foil." In a Prussian police ordinance for May, 1857, the public are warned of the danger of purchasing snuff packed in leaden wrappers. It is there stated that several cases of lead-paralysis have occurred from the use of this snuff, as it is, under these circumstances, frequently impregnated with lead. (Casper's *Vierteljahrsschrift*, Jan. 1858, pp. 184 and 163.) Dr. Sonnenkalb, of Leipsic, considers that snuff frequently acquires an accidental impregnation of lead by reason of the coverings of lead in which it is packed. He has collected nineteen cases of this form of chronic poisoning; in fourteen of these there was paralysis, and in five there were symptoms of gastric disturbance. The arms were most commonly affected with paralysis in the extensor muscles, which wasted. In twelve cases there was a blue color of the gums. All suffered from colicky pains and constipation. The poisoned snuff had been used for a period of from six months to twenty years, and on leaving it off the patients improved rapidly, and eventually recovered. (See also a paper by Dr. Garrod, *Lancet*, Dec. 1870, p. 781, and *Pharm. Jour.*, 1870, p. 465; and another by Dr. Flinzer, Horn's *Vierteljahrsschrift*, 1868, vol. 2, p. 175.)

Chocolate is also sold in wrappers of this kind. An important case was tried at the Guildhall Summer Sittings, 1857 (*Adnam* v. *Betts*), which, for the first time, revealed an extensive source of lead-poisoning specially adapted for the infant population of this country. The plaintiff, who was a manufacturer of *groats* as an article of food for children and invalids, claimed damages of the defendant on the ground that the food had been damaged, and rendered noxious and unsalable by reason of its having been wrapped in a spurious metal, consisting chiefly of lead, but sold to the plaintiff as tin-foil. Mr. Brande, Mr. Scanlan, and myself examined many packets of the food, which had been sent to the colonies and returned as damaged. We found that the metal wrappers were extensively corroded in a number of small holes; the layer of the food in contact with the metal was discolored and strongly impregnated with lead. On examining the metallic wrapper, sold as Betts's patent metal, or tin-foil, we found it to consist of from seventy to eighty parts of lead, and of twenty to thirty parts of tin. The tin gave merely a facing to the lead and made it appear like tin-foil. The plaintiff lost his case chiefly on the ground of his having purchased the metal at a price at which he ought to have known pure tin-foil could not be sold. Assuming this to be a good answer in law, it is fair to question the propriety of a patent being legally granted for the sale under a false name of such a spurious and noxious alloy as this. It is sold for wrappers and capsules, and is placed in contact with liquids and solids used as articles of food; it is therefore liable to impart to them a dangerous impregnation of lead. If a man sells copper faced with gold as patent gold, he may be punished for fraud, although the damage is here only of a pecuniary nature. The sale of *lead-foil* as tin is legally licensed. It has not yet been made a subject for a penalty under the Adulteration Act.

An alloy of lead and tin was employed, a few years since, in the form of a screw-capsule, as a patented substitute for corks in bottling wines, preserves, etc. On examining the preserved fruits, and the vinous liquids kept in bottles thus stopped by patented noxious stoppers, I found them to be strongly impregnated with lead! In France, the sale of this article as tin or tin-foil carries with it certain penalties. In a recent case, proceedings were taken by a purchaser against the vendor. It was proved that the so-called tin-foil contained a large quantity of lead; and the vendor was summarily condemned to a month's imprisonment, 150 francs fine, and costs, as well as a return of the money paid for the metal, which was ordered to be confiscated by the Court. (*Journal de Chimie*, Jan. 1858, p. 50.) In England the rule is *caveat emptor*.

CHAPTER XLIV.

POISONING WITH LEAD—LEAD IN AERATED WATERS—CHRONIC POISONING—LEAD IN PURE WATER—EFFECT ON RIVER AND SPRING WATER—PREVENTIVE SALTS—POISONING OF CATTLE WITH LEAD.

LEAD IN WATER.

ARTIFICIAL AERATED WATERS are now sold in the convenient form of bottles provided with stopcocks made of pewter containing much lead. Some of the gazogenes are of this description, and, by long use or contact, the water contained in them acquires an impregnation of lead. Where block-tin is used with but little lead, the water may be safely taken, a slight solution of tin not rendering the water noxious.

Dr. Attfield states that he has examined the aerated waters contained in siphon vases, and that he has in some cases found them to contain tin, but not lead. The tin is derived from the pewter taps. (*Brit. Med. Journ.*, June 6, 1874.) Another chemist states that he has found lead dissolved from the pewter taps of the siphon vases, and that both lead and tin are contained in these carbonated waters. (*Brit. Med. Journ.*, June 13, 1874, p. 789.) In some samples of these waters I have detected traces of lead, and in others none. When well-tinned siphon taps are used there is no danger of impregnation with lead, but the leaden taps and pipes of the common gazogenes contain much lead, and are liable to give a noxious impregnation of lead to the carbonated waters contained in them, and to lay a foundation for chronic lead-poisoning.

This alloy is largely manufactured into capsules for covering the corks of bottles of wine. If the cork is at all porous, or fits badly, the acid of the wine acts upon the alloy, producing carbonate of lead round the neck of the bottle, and a layer of the same compound on the inside of the capsule. In examining the capsules and corks after the bottles have been lying some months in a cellar, the former show marks of corrosion, and when the white substance is treated with iodide of potassium and acetic acid, the presence of lead is indicated by the production of the yellow compound of iodide of lead. In one capsule I found a deposit of from two to three grains of white lead. Some portion had even penetrated into the substance of the cork. This spurious foil may be thus detected: Add to two ounces of water one drachm of sulphuric acid and one drachm and a half of nitric acid. Plunge a slip of the foil into this mixture. The tin is soon oxidized and removed, and the lead appears under its true color and with its usual properties. It will be thus seen that it constitutes the great bulk of the alloy.

Dr. Milne, of Glasgow, examined various samples of soda-water as ordinarily sold, and found lead in several of them, varying in proportion from one-tenth to six-tenths of a grain in a gallon; and in aerated lemonade from two-tenths to four-tenths of a grain. (*Pharm. Journ.*, Oct. 3, 1873, p. 261; and Jan. 23, 1875, p. 583.)

It would appear from a case lately published by Mr. Wilson (*Brit. Med. Jour.*, Sept. 5, 1874, p. 323) that the manufacturers of soda-water in glass bottles, are not sufficiently careful in the selection of the water employed for impregnation with carbonic acid. One of his patients suffered from all the usual symptoms of chronic lead-poisoning, and after some time the lead was traced to, and discovered in, the soda-water, of which she had been in the habit of taking daily six or seven bottles. The water, as is frequently the case, contained no carbonate of soda, but only carbonic acid; and lead was found in it in the proportion of nine-tenths of a grain in a gallon. It is probable that the carbonic acid, to some slight extent, aided the solubility of the lead as carbonate. The manufacturers must have used water already containing lead derived from leaden pipes or cisterns.

Water.—Of all articles of diet, there is none which has been so fruitful a source of lead-poisoning as water. The symptoms and appearances have been elsewhere described (*ante*, pp. 402–404). It will now only be necessary to consider the circumstances under which water, distributed in leaden pipes, or stored in leaden cisterns, may acquire a poisonous impregnation: 1. Absolutely pure water, recently boiled to deprive it of air, and placed in contact with a clean surface of lead in a hermetically sealed tube, has no chemical action on the metal. 2. The same water, exposed to air, produces in a few minutes around the surface of the metal a milky-looking film; and, in twenty-four hours, this shows itself as a white compound, diffused in pearly scales either loosely adhering to the lead, or as a white sediment at the bottom of the vessel. The compound thus formed is a mixture of hydrated oxide and carbonate of lead, the carbonic acid and oxygen being derived from the air. This compound is not dissolved in the water to any perceptible extent, but is mechanically diffused through it. Water in this state is, however, just as dangerous to health as if the lead were dissolved. The more nearly pure, or the more free from saline matter the water is, the more intense is this chemical action; and it commonly proceeds until all the lead is converted into this chemical compound, or until the surface of the metal is invested with so closely adhering a film, that neither oxygen nor carbonic acid can reach it. Water thus contaminated, if passed through a good sand and charcoal filter, will lose the whole of the salt of lead, and be rendered innocuous. 3. *Rain*, *snow*, and *ice water* (Wenham ice), being generally remarkably free from saline matter, act in a similar manner upon lead. An epidemic of lead-colic, which appeared many years since, at Amsterdam, was traced to the use of rain water collected from leaden roofs. (Christison on *Poisons*, p. 526.) Rain-water which has passed over slate or tile does not, however, readily act upon lead. 4. *Pure*, or *soft spring*, or *lake* water, containing only a few grains of saline matter to the imperial gallon, has been hitherto considered as dangerous for use. The water supplied to Tunbridge, in 1815, through leaden pipes, was what is called a pure water, and its use caused an outbreak of lead-colic in that town. The water of Claremont, containing only five grains of saline matter to the imperial gallon, produced, in a few months, a severe form of lead-colic (*ante*, p. 402) in the royal family of France, elsewhere described. The water of the Surrey sands has an evil reputation in this respect: it is comparatively pure, and, generally speaking, acts strongly on lead. The severe cases of lead-colic met with by Dr. Thomson (*ante*, p. 401) were traced by him to the Surrey sands' water. The chief part of the saline matter is common salt. The water supplied through leaden pipes to the royal kennels, at Ascot, caused, a few years since, a general lameness (from lead-paralysis) among the hounds. In that neighborhood I have found one sample of water to act powerfully on lead, and another not, although both would come under the head of pure waters.

In the year 1854, the influence of *Lake water* on lead underwent a close scrutiny before a Committee of the House of Commons, in reference to the proposed supply of Glasgow with the pure water of Loch Katrine, containing only two grains of saline matter to the imperial gallon. I found this water to act strongly on lead when the bright and highly-polished metal was immersed in it under a free exposure to air. It had no action on lead when the metal was in its ordinarily dull state. When this water was allowed to stand some time over masses of limestone and old red sandstone, its chemical action on lead ceased. The question of the safety of supplying such a water as this through leaden pipes to a large and populous city, gave rise to a great difference of opinion among a large body of scientific chemists, who were examined before the Committee. The question, however, was finally decided by an appeal to the experience of other towns in which a water of similar quality had been many years in use. Inverness had been supplied from Loch Ness, through leaden pipes, with water as pure as that of Loch Katrine; and during a period of twenty-four years there had been no cases of lead-colic in that town from the use of this water. A remarkable fact transpired with respect to the Ness water. A portion drawn from a leaden pipe in a private house, at Inverness, contained no lead; but when the water was placed in contact with a bright surface of the metal, it rapidly acted on it. The town of Whitehaven, which is supplied from Enerdale Lake, presented another instance of the distribution of pure lake water through lead, without any accident occurring from its use, in a large population. Yet the water of this lake exerts a chemical action on bright lead. On these practical grounds and proved results, Glasgow was permitted to receive its supply from Loch Katrine. There is no doubt that, in these cases of non-contamination, the leaden pipe or cistern soon acquires a closely adhering deposit, which is sufficiently thick to prevent any further chemical action of the water on the lead beneath. Twenty years have passed, and, so far as I can ascertain, there have been no instances of lead-colic produced by this water. The surface of the interior of the leaden pipes probably acquired after a short time a protective coating.

The facts above mentioned show that, whatever scientific theories may exist regarding the poisoning of water by lead—the question cannot be strictly determined from the known constitution of the water; it should be always based on actual experiment. For ordinary purposes I find it sufficient to employ eight ounces of the water in an open beaker, plunging into it a portion of plumber's sheet lead, four inches long by one inch wide. This represents a square inch of metallic surface to one ounce of water. The lead should be used in the state in which it is ordinarily employed for cisterns by the plumber (six pound lead). A portion of the lead, exposing about sixteen or twenty square inches, should be immersed in twenty ounces of water. The chemical change, if any, should be daily noticed, and the water tested by a current of sulphuretted hydrogen gas in a glass tube, about twelve inches long and one inch in diameter. By looking down the length of the tube, after the gas has been passed into it for a sufficient time to impart a smell, the slightest change to a brown color will be immediately perceptible. The quantity present in a given sample may be estimated by passing the gas into similar tubes containing minute but decreasing fractional proportions of a grain of lead in a gallon of water. A comparison of the shade of color with the shades of the standard tubes, will thus enable the operator to fix the proportion of lead with sufficient accuracy for practical purposes.

It is necessary to bear in mind that a water may contain a noxious proportion of lead, but present no opacity; the lead may not be diffused as insoluble carbonate, but be *dissolved* in it as chloride or nitrate. In this case it may be tested in the entire state; and if this should fail to reveal the presence of lead, half a gallon of the water may be evaporated, and, towards the end of the operation, the residue may be dissolved in diluted nitric acid, and then tested. The quantity of lead present in water which is noxious to health, is usually very small. In the Claremont case, Dr. Hofmann found only one grain of lead in a gallon; but a smaller portion would, in time, be productive of serious injury to health. (*Med. Gaz.*, vol. 44, p. 261.) This is perhaps the smallest quantity of lead in water accurately recorded to have produced the effects of poisoning. No symptoms appeared until after the water had been in use for a period of from five to seven months, and more than half of those who used the water escaped any ill effects. According to the late Mr. Herapath, the symptoms of lead-poisoning have been produced in a community by so small a quantity as one-ninth of a grain of lead in a gallon; and Dr. J. Smith, of Aberdeen, concludes from his investigations that the limit of manifestly deleterious action would seem to be somewhere between one-tenth and one-twentieth of a grain in a gallon. (Dr. Penny, *Report on Loch Katrine Water*, p. 107.) Waters collected from lead-mine districts generally contain lead either in suspension or in solution. In one of these proposed to be supplied to Wrexham, in North Wales, I found the proportion of lead to be one-eleventh of a grain in a gallon—a quantity which might prove noxious to some portion of a town population, as a result of long-continued use. A medical officer of health deposed before a committee of the House of Lords, in May, 1864, that there was less than one-fourteenth of a grain of lead in a gallon, and that this proportion, if present, would do no injury, while one-twelfth of a grain would be noxious, although the data upon which this sharply defined distinction was based were not made public! According to Sir R. Christison, all waters act more or less upon lead, and he assigns, as a limit of safety for persons using a water for domestic purposes, a proportion of lead not exceeding one-millionth part, or about one grain in fifteen gallons of water. (*Pharm. Journ.*, April, 1872, p. 852.) A water which acts chemically on the metal may soon cease to act by reason of a hard deposit taking place in the interior of the pipe or cistern, and the metal is thus protected from further chemical action. When the water, before entering the pipes, is already provided with so much lead that it easily admits of detection in a pint, a slight additional impregnation will suffice to render it poisonous. A safe sanitary rule would be that suggested by the late Dr. Penny. All lead-contamination is objectionable, and no degree of it can be considered safe. Lead is an accumulative poison, and affects some persons powerfully in the smallest quantities. An evil practice has lately sprung up of substituting for pure block tin a cheap alloy of tin and lead, in the so-called tinning of iron and copper utensils. The small supply-boilers of cisterns, supposed to be tinned, are really covered with a layer of pewter; and lead may thus be conveyed into food and water under circumstances not suspected. Mr. Gobley has fully pointed out the danger of this practice in reference to public health. (*Ann. d'Hyg.*, 1869, vol. 1, p. 237.)

Dr. Earle was called to a family said to be suffering from cholera. He found six men stretched on the floor of a room vomiting and suffering from severe pain in the abdomen. The pulse was slow and feeble, and the bowels were constipated. On inquiry, it was found that the men had been drinking water from a teakettle, in which lead had been melted about ten days before. They had used it daily with hot water. On the next morning two women were attacked with similar symptoms. Under treatment, they all recovered. (*Amer. Jour. Med. Sci.*, Jan. 1874.)

The quality of the *lead* appears to exert some influence on the production of these chemical changes. Certain kinds of lead are speedily acted on and corroded by water; others with difficulty. In the experiments on Loch Katrine water, the late Dr. Miller and I found that the lead supplied for small cisterns, obtained from different parts of London, when used in the state in which it was received, produced no chemical changes with the water; whereas Dr. Penny found that the lead supplied to him for a similar purpose, in Glasgow, was rapidly attacked and corroded. The cause of these differences is not at all understood; but one fact is certainly established,—the more highly polished and the brighter the lead, the greater is the probability that, *cœteris paribus*, water will be rendered noxious by contact with it under exposure to air. An alloy of lead with tin retards, but does not prevent this chemical action.

5. *River Water—Hard Spring Water.*—River and spring waters, containing a moderate amount of saline matter (twenty to forty grains in the gallon), do not, in general, give rise to a noxious chemical action on lead, provided they contain sulphate and carbonate of lime, and not too large a proportion of alkaline chlorides or nitrates. Distilled water acting powerfully on lead is deprived of this chemical action when a solution of sulphate of lime, or of carbonate of lime in carbonated water, is added to it. The quantity need not be such as to impart to the water any remarkable taste, and there is reason to believe that these calcareous compounds dissolved out of the earth, confer on river waters in the south of England the immunity which they are known to possess in reference to an action on lead. The Thames River water, containing about seventeen grains of saline matter in the gallon, is remarkably free from this action; and when it is considered that it is supplied, through lead, to the extent of 110,000,000 of gallons per diem, to a population exceeding three millions, and that cases of lead-colic from the use of London water are unheard of—it is obvious that it is so constituted as to prevent these dangerous chemical changes. The principal salts in this water are carbonate and sulphate of lime, with a small proportion of common salt. Thames water frequently contains alkaline nitrates, derived from the decomposition of nitrogenous matter, and these salts may be the means of giving to it, occasionally, an impregnation of lead. The alkaline chlorides also favor the formation of chloride of lead, which the water may hold dissolved; and, at the same time, they do not prevent the production of the noxious carbonate. Chloride of sodium appears to be the chief ingredient in the Surrey sands water. The Claremont water contained five grains of saline matter in the gallon, of which one-half was common salt. In the Surrey sands water, which caused the symptoms in Dr. Thomson's cases, the quantity of mineral matter amounted to about five grains and a half in a gallon, and four-fifths of this were common salt. Some kinds of spring water, containing a large proportion of sulphate of lime and other salts, are found to act powerfully on lead. Mr. Osborne considers that lead, in the form of chloride, is liable to be produced in the spring water of the wells around Southampton. (*Pharm. Times*, September 26, 1846, p. 64.) Artesian water, by its alkaline salts and the large proportion of neutral salts contained in it, is found to act upon lead. In short, both hard and soft water may be rendered noxious by contact with lead, according to circumstances but little understood. All kinds of theories have been put forward to account for these chemical changes; but none are satisfactory, and none will explain all the facts. An excess of saline matter in some cases, and a total deficiency of it in other cases, may equally produce noxious effects. Sulphate and carbonate of lime—which, in small proportion, appear to be beneficial—are injurious when in large quantity; and the alkaline nitrates and chlorides appear to be injurious under all circumstances. It is a curious fact, that lead may be kept immersed in a sample of

water, unchanged; while the vapor of that water, as it rises (distilled water), will corrode a leaden cover placed over the vessel, and thus impregnate the water below. As a general rule, soft and pure waters act upon lead, and hard waters do not; but a water must not be condemned as dangerous because it is soft, nor approved as safe because it is hard. In reference to some soft waters, the chemical action soon ceases; while in others it appears to continue unchanged so long as there is any metal under free exposure to air; but this rule is not so fixed as to dispense with the necessity of a special examination in each case. The variable intermixture and proportion of salts and gases contained in natural waters, render it impossible to state, *a priori*, what will be the results of experiment.

When a water continues to act powerfully on lead and iron, and there are no means of changing the supply, it would be proper to substitute well-tinned pipe, glass, or earthenware, for lead. A cistern of slate should also be substituted for one of lead. For drinking purposes, the water should always be filtered through a bed of sand, as this effectually separates the insoluble carbonate of lead. These rules should be adopted when there is a tendency to act upon lead, whether the water be soft or hard.

Poisoning of Cattle with Lead.—Medical evidence has of late years been required in cases in which damages have been claimed for alleged loss of cattle by reason of the vicinity of lead-works. I have elsewhere referred to two cases, one in which a mare died from drinking at a stream, into which carbonate of lead had been discharged from certain lead-works. Dr. Wilson conducted the examination in this case. The cause of death was clearly traced to lead, and a quantity of this metal was separated from the viscera of the animal. Lead was found in the water of the stream, and in the vegetables growing on the soil. (*Ed. Monthly Journal*, 1852, vol. 14, p. 386.) In the case of *Stephens* v. *Barwell* (Wells Autumn Assizes, 1855), it was alleged on the part of the plaintiff that a large number of sheep and cattle had been destroyed by fumes of lead escaping from a chimney on the defendant's works. The case involved this curious point, namely: admitting the sheep and cattle to have been destroyed by lead (of which there was not much doubt, at least in some instances), whether the lead was deposited on the herbage from the defendant's chimney, or taken up by the plants from the soil, and incorporated with their tissues. Mr. Brande and I were required to examine the defendant's flue, in the Mendip Hills, and found, from its enormous length (upwards of 2000 feet), and the constant cooling and washing of the vapors which traversed it with a large quantity of water, that every reasonable precaution had been taken to prevent their escape. In going over the plaintiff's ground, we could not perceive on the herbage, far or near, the slightest appearance of a deposit of white lead, or of lead in any form. We found, however, that the soil and the herbage in the fields where the cattle had been pastured, was impregnated with lead, and we also found by experiment that seeds sown in the leaden soil brought for this purpose to London, produced plants which contained lead in their tissues, and that this was probably the cause of lead-poisoning among the cattle. The soil had derived the lead from the disintegrated slag of ancient mineral workings. It contained silicate and carbonate of lead amounting to as much as two per cent. These facts were stated on the part of the defendants, and they further alleged that cattle had died from lead-poisoning on this farm before their works were in operation; that they had died from the same cause in spots remote from these works, wherever the pasture of the district happened to be over lead-slag, so that some localities had acquired an evil name from this circumstance, and the pasturing of cattle in such places had been avoided; that there was a sufficient cause of lead-poisoning in the herbage grown on the soil, which abounded in lead, and in the water which the cattle drank; and there was a total absence of proof of any deposit of lead from a flue on the soil or the surrounding vegetation. Upon this statement, and without any evidence being called for, an arrangement was made between the parties. Sometimes this form of poisoning is the result of accident. Dr. Gordon, of Aberdeen, lost seven head of fine cattle, which were grazing in a field. On opening the stomachs, white lead was found therein. The deaths here were traced to some refuse paint or white lead, which had been carelessly left about as waste. The sudden deaths of animals may be frequently due to a cause of this nature, and suspicion fall unjustly upon parties who have the care of them. Poisonous materials of this kind should be effectually disposed of and not thrown into ash-pits or on waste land. There are other forms of lead-poisoning to which cattle are subject (*ante*, p. 391).

CHAPTER XLV.

COPPER—EFFECTS PRODUCED BY THE METAL AND ITS ALLOYS—BLUE VITRIOL—VERDIGRIS AND OTHER SALTS—SYMPTOMS—ACUTE AND CHRONIC POISONING—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—TREATMENT—CHEMICAL ANALYSIS—TESTS—COPPER IN ORGANIC LIQUIDS—IN THE TISSUES—IN THE EARTH OF CEMETERIES—IN ARTICLES OF FOOD.

COPPER.

General Remarks.—Copper itself is said to be destitute of poisonous properties; but it would appear that when alloyed with other metals and reduced to a finely pulverulent state, it may act as a poison. A singular instance of this kind is on record. The printing in gold, as it is termed, is performed by means of a species of brass or copper alloy. The letters are printed with a mixture of size and gamboge; and the copper alloy, reduced to such a fine state of division that it floats in the atmosphere in an impalpable dust, is then brushed over the surface. A boy employed in this occupation was, on the third day, seized with vomiting of a green-colored fluid, heat and constriction of the gullet, pain in the stomach, loss of appetite and rest, and a severe itching in all those parts which were covered with hair. These on examination were found to be of a deep green color. The boy soon recovered. About twelve other persons, employed in the same work, suffered from similar symptoms; but this did not prevent them from continuing the work. The poison in this case probably entered the system through the lungs and skin. This peculiar effect of finely divided copper in giving a green tint to those parts covered with hair, is mentioned by Dr. Falconer in his *Essay on the Poison of Copper*, p. 42, published in 1774. Dr. Clapton has pointed out another symptom, namely, a green line on the margin of the gums. He met with this in a sailor, and in some working coppersmiths. (*Med. Times and Gaz.*, June, 1868, p. 658.) Two of these cases I saw in 1868. The green line was well marked. The men brought with them a hammer used in their work. It had a greenish color, and this was shown by tests to be owing to copper. The perspiration from the hands in working had converted the copper into subchloride, and thus led to its absorption by the skin. Several cases of chronic poisoning by copper among coppersmiths, have been treated by Dr. Cameron, of Liverpool, but this symptom was not noticed. (*Med. Times and Gaz.*, 1870, vol. 1, p. 581.)

An alloy of copper, made to resemble gold (Dutch gold), is largely used in the ornamenting of gingerbread and confectionery. I am not aware of any accident having occurred from its being thus eaten; but it is a noxious practice, and in France is especially prohibited, under a penalty, by police regulations. (*Journal de Chimie*, Février, 1847.) This alloy is easily known from gold by its entire solubility in nitric acid, with which it forms a greenish-colored solution of nitrate of copper. When metallic copper is swallowed, colicky pains and other symptoms sometimes follow in consequence of the metal becoming partially oxidized and dissolved. The experimental researches of M. Leportier show that the pure metal is not directly poisonous (*Ann. d'Hyg.*, 1840, vol. 2, p. 99); but it may cause death as a mechanical irritant. Copper coins when swallowed may produce a certain amount of poisonous salt from the action of the alkaline chlorides in the stomach; but the great danger to be apprehended in these cases is that they are liable to cause death by a mechanical action.

All the salts of copper are poisonous. The two most commonly known are the *sulphate*, *blue vitriol*, or *blue stone*, and the *subacetate* or *verdigris*. These substances have been frequently taken and administered in large doses for the purpose of suicide and in attempts at abortion and murder. In the latter case the attempt has been immediately discovered, owing to the strong metallic taste as well as color possessed by the salts. The taste would in general render it impossible that a poisonous dose of blue vitriol or verdigris should be taken unknowingly. With the exception of the wilful use of these salts, poisoning with copper is commonly the accidental result of the use of this metal for culinary purposes.

Symptoms—Acute Poisoning.—Poisoning with copper may be divided into the acute and chronic forms. Cases of acute poisoning from blue vitriol or verdigris are occasionally met with. The symptoms have nearly the same character and course in reference to these and all the other compounds of copper, if we except the arsenite, which has been already considered among the arsenical poisons (*ante*, p. 337). When the sulphate is taken in doses of half an ounce or upwards, a strong metallic taste is perceived in the mouth ; there is constriction in the throat and gullet, with griping or colicky pains in the stomach and bowels, increased flow of saliva, nausea, and vomiting. Blue vitriol is a powerful emetic, and vomiting is rapidly excited by it. The abdomen is distended, the pain in this cavity is increased by pressure, and not relieved by vomiting, and there is purging with tenesmus. The vomited liquids have a bluish or greenish color, and the discharges by the bowels are sometimes greenish, bloody-looking, or dark-colored. These symptoms commence generally in a few minutes after the poison has been taken, and are fully developed within one or two hours. Jaundice and suppression of urine have been observed in some cases. The abovementioned symptoms are chiefly connected with the *irritant* effects of the salt of copper on the stomach and bowels. When the poison has been absorbed, another set of symptoms, indicative of an action on the brain and nervous system are witnessed. There is hurried and difficult breathing, with a small quick pulse, great weakness, intense thirst, cold perspiration and coldness of the limbs, headache, giddiness, stupor, coma, tetanic or other convulsions, followed by paralysis of motion or sensation in the limbs. The patient gradually sinks, and dies exhausted in a few hours or days. In some cases the symptoms assume at once an entirely nervous character: there are severe headache, great depression, restlessness, trembling of the limbs, cramps, coldness of the surface, small irregular pulse, dilatation of the pupils, with stupor, coma, tetanus or paralysis. These symptoms are, however, commonly preceded by vomiting, purging, and colicky pains in the abdomen.

In forming an opinion from the green color of the vomited matters in alleged cases of poisoning by copper, the practitioner must remember that a morbid state of the bile may give a most vivid copper-green color to liquids thrown from the stomach. I have seen this in one case, and from the intensity and persistency of the green color, poisoning was suspected. A slight chemical examination will show whether the color is owing to bile or to a cupreous poison. (See *post*, Analysis.)

Verdigris produces symptoms somewhat similar to those caused by the sulphate of copper. There is a strong styptic metallic taste, with a sense of constriction in the throat, followed by severe colicky pains, vomiting of a green-colored liquid, purging, and tenesmus. In a case reported by Pyl, a woman who took *two ounces* of verdigris died in three days. In addition to the symptoms above described, there were convulsions and paralysis before death. Niemann relates that a woman, æt. 24, swallowed *half an ounce* of verdigris, and died under symptoms of violent gastric irritation in sixty hours. (*Taschenbuch*, p. 458.) In consequence of the great uncertainty of its operation, this compound is not employed as a medicine.

A case of poisoning by this substance, in which the symptoms were accurately observed, is reported in the *Edinburgh Medical and Surgical*

Journal, for July, 1844. A woman, æt. 28, swallowed a large dose of verdigris. She was soon afterwards seized with great anxiety, vomiting, acute pains and swelling of the abdomen, sensation of burning heat in the throat, coldness, and severe cramp in the extremities, a laboring pulse, swelling of the face, with the eyes sparkling. An emetic brought away some half-digested food, without any traces of poison. The next morning there was pain in swallowing, swelling of the throat, the abdomen distended, and painful on the least pressure, the countenance heavy, the face flushed, and the pulse oppressed. About two pounds of a distinctly greenish fluid, with some blood, were thrown off the stomach. The symptoms became aggravated; the face and eyelids swollen and red, the eyes prominent, the abdomen drawn in, and the rectum irritable and painful. On the second day there was a tendency to coma, the face was pale, the lips swollen, the gums ulcerated, and there was an abundant discharge of viscid saliva. Purging took place for the first time since the poison was taken; and acetate of copper was detected in the discharges in rather large quantity. There were several spasmodic fits. On the third day some viscid glairy matter, of a greenish color and tinged with blood, was vomited, and the spasms continued. On the fourth day bleeding from the nose with general cramps came on, and the urine was suppressed. There was coldness of the surface with convulsions. After the lapse of about a week the patient still had vomitings of greenish glairy matters, with uneasiness in the abdomen; but from this date she gradually recovered.

The subchloride or *oxychloride* has thus given rise to accidental poisoning. This compound is used as a green pigment. The following is a case of poisoning by it: A boy, between two and three years of age, swallowed part of a small cake of green water-color, such as is sold in the color-boxes for children. Very soon afterwards he was attacked with vomiting and coldness of the extremities. Notwithstanding the exhibition of an antimonial emetic, the symptoms continued to become aggravated, and the child died. (Henke's *Zeitschrift der S. A.*, vol. 1, p. 188, 1844.) This salt of copper is often formed in culinary utensils, and may then give rise to accidents, when food containing common salt has been prepared in the copper vessel without proper precautions. (See *Journal de Pharmacie*, Juin, 1845, p. 471.) Professor Barzellotti relates an instance in which he himself narrowly escaped partaking of the poisonous food. At a monastery near Sienna the monks were one day, soon after dinner, seized with violent symptoms of irritant poisoning. They suffered chiefly from severe pain in the abdomen, nausea, difficulty of passing urine, spasms of the muscles, and trembling of the limbs. Those who were affected with vomiting and purging were speedily relieved; but others, who had no evacuations, suffered from giddiness, headache, intense thirst, and an unpleasant metallic taste in the mouth. Remedies were applied, and they all eventually recovered. It appeared, on inquiry, that the monks were in the habit of keeping their salt fish in a copper vessel, in which it was dressed for a second day's meal. This vessel was badly tinned; and when the fish was examined, it was observed to be covered with a green jelly, and the sides of the vessel with which the fish was in contact had a green color. The cause of the symptoms was no longer doubtful; subchloride of copper had been here formed by the action of the salt on the metal. (*Quest. di Med. Leg.*, tomo 2, p. 185.) Several cases of a similar kind are reported by Orfila, vol. 1, p. 619.

A case of poisoning by the *carbonate of copper* occurred to M. Desgranges, of Bordeaux. A man died in about six hours, as it was supposed from the effects of an unknown quantity of this poison which he had taken. When first seen he was insensible; he had sustained some violence from a fall, and there was great coldness of the extremities. There was neither vomiting, purging, nor pain in the abdomen on pressure. (*Med. Gaz.*, vol. 31, p. 495.)

Chronic Poisoning by Copper—Copper Colic.—When the symptoms of acute poisoning have passed away, when the cupreous salt has been taken for a long period in small doses, or the person has been exposed to emanations from copper salts or alloys, other effects are manifested. The most prominent after-effects are excessive irritability of the alimentary canal, attended with frequent disposition to vomit—colic, purging, and tenesmus; and there is at the same time loss of appetite, alternations of cold and heat, great prostration of strength, with emaciation, tremors of the limbs, and occasionally paralysis. There is a coppery or metallic taste in the mouth, increased thirst, hot skin, with a small, frequent, irregular pulse. After a few days, there is tenderness with distension of the abdomen, and colicky pains of a severe kind, with symptoms resembling those of dysentery; the evacuations are of a greenish color, and mixed with mucus and blood. There is jaundice, with some of the nervous symptoms already described under the head of acute poisoning. The patient sometimes dies from fever and exhaustion. (For a further account of the symptoms, and those which accompany copper-colic, see Böcker, *Vergiftungen*, 1857, p. 42.)

One of the most complete accounts of chronic or slow poisoning by copper, has been published by Dr. Moore. It occurred from want of cleanliness in the use of culinary utensils, and it shows that, without circumspection, a medical man may be completely deceived respecting the origin of the malady affecting many persons simultaneously. On the return of the Indian Coolie emigrants from Guiana to Calcutta, a kind of acute idiopathic dysentery made its appearance in the ship, and it was at first referred to bad water, change of climate, and other causes. Dr. Moore examined the copper-plates on which the fish, rice, and ghee (butter), eaten by the natives, was cooked, and found the surface was coated with a green composition, which, when scraped off and examined, proved to be a mixture of chloride and sulphate (?) of copper. The cause of the disease was then apparent. A few hours after taking the meal, the patients complained of violent pains and cramps in the stomach and lower bowels, and there was constant vomiting of greenish and yellowish-green bile. When this was not ejected from the stomach, their sufferings from dry-retching were most severe, and the feeling of constriction in the lower part of the chest and along the course of the gullet was still more distressing. Every twenty minutes there was an attempt to evacuate the bowels, but no feculent matter was discharged; blood in small quantities, slimy mucous stools, tinged with blood, shreds of lymph, and frothy ash-colored secretions, were passed from the rectum without affording the patients the slightest relief. Pressure over the abdomen, especially in the pit of the stomach, and in one case over the arch of the colon, caused severe pain. There were griping pains in the loins and sacrum, at the navel, and in the iliac region, with tenesmus and a burning sensation at the anus. In the commencement of the attack, there was acute fever, pungent heat of the skin, headache, urgent thirst, loss of appetite, prostration of strength, furred and clammy tongue, foul taste in the mouth, with a rapid, small, and wiry pulse. In the more severe cases, there was great depression of the vital powers, the pulse exceedingly rapid and weak, the skin cold, extremities benumbed; the secretion of urine was in a few instances suppressed, in others the urine was retained in the bladder. The symptoms in most instances subsided in eight or ten days under the free use of emetics and castor oil; in others, a long time elapsed before the mucous discharges from the alimentary canal and the tenesmus abated—the disease assuming all the characters of chronic dysentery. One man was subsequently attacked with symptoms of chronic poisoning in an aggravated form, from neglect in the use of a copper vessel, and sank under the attack. (*Lancet*, April 11th, 1846, p. 414.) Dr. Moore considers that the attacks of cholera and of acute or chronic dysentery, under which Europeans arriving in the East Indies so frequently suffer, are in many cases due to the general employment of copper utensils for culinary purposes, and from the want of cleanliness on the part of the native cooks, who use butter, salt, and acids, without removing the cupreous incrustation which is formed on the surface, or in the rims of the vessel. Hot butter or lard, like hot oil, readily dissolves copper, forming fatty salts, of which oxide of copper is the base.

French pathologists have described a copper colic to which workers in this metal are liable, owing, as it is supposed, to the inhalation of the fine dust of copper or its oxide. According to Orfila, it is in some respects analogous to lead colic, but it differs from it in being accompanied by a greater degree of irritation in the stomach and bowels. (*Toxicologie*, vol. 1, p. 912.) The existence of this as an independent form of colic has been denied by some authorities. (*Annales d'Hygiène*, 1847, vol. 1, p. 392; and Avril, 1858, p. 328. Casper's *Vierteljahrsschrift*, 1852, vol. 1, p. 79; 1855, vol. 2, p. 222; 1856, vol. 2, p. 41; and 1857, vol. 2, p. 228.) There is, however, sufficient evidence to establish the existence of this form of copper-poisoning. Dr. Corrigan, who has given some attention to this subject, has arrived at the following conclusions: 1. Copper will act as a slow poison, by absorption, undermining the constitution, producing emaciation, catarrh, and loss of strength, and leaving the system in a state little capable of resisting the ordinary exciting causes of many diseases. 2. The symptoms, although not acute, are well marked: they are emaciation, a cachectic appearance, loss of muscular strength, colicky pains, cough, without physical signs to account for it, and the peculiar characteristic signs of retraction of the gums, with a purple, not a blue edge. 3. In none of the cases detailed, although there was muscular debility, was there either acute colic with constipation, or the local paralysis that so often results from the poison of lead; and the color of the gums was quite distinct from that produced by lead. 4. Copper, in chronic poisoning, seems to exert its deleterious influence mainly on the nutritive functions, or assimilation, including absorption and secretion, while lead acts energetically on the nervous system of both organic and animal life, exhibited in its action on the former by producing obstinate constipation, and on the latter by the violent pains of lead colic, as well as by the production of a peculiar form of paralysis. (*Dublin Hospital Gazette*, for September, 1854; *Lancet*, January, 1855.)

Effects of External Application.—The salts of copper are capable of acting locally, and if applied to a wounded or ulcerated surface, they may become absorbed, and thus affect the system. Sulphate of copper is occasionally used as an escharotic. The solution of this salt, after frequent contact, hardens the unbroken skin, discolors it, and impairs its sensibility. Orfila found that two drachms of acetate of copper, finely powdered, when introduced beneath the cellular membrane of the neck of a large dog, caused death in five days. In another experiment, the same dose, applied to the cellular tissue of the thigh, killed the animal in thirty hours. (*Tox.*, vol. 1, p. 618.) Violent phlegmonous inflammation is sometimes occasioned by small quantities of the salts of copper becoming introduced into the system through wounded or abraded surfaces. Mr. Stafford met with a case in which a woman pricked her thumb with a pin. She afterwards scoured out a dirty copper vessel, and her thumb immediately swelled to double its natural size. The whole hand and arm became much swollen and inflamed, and extensive abscesses formed: the patient also suffered from fever, from which she slowly recovered. A second case occurred to the same gentleman, in which severe symptoms followed a puncture produced by corroded copper wire. (*Med. Gaz.*, vol. 35, p. 828.) In these cases the poisonous salt may be the carbonate, subacetate, or subchloride—most commonly the former. It is probable that the severity of the symptoms may be in some instances ascribed to peculiarity of constitution, the very small quantity of the salt of copper which can be absorbed scarcely sufficing to account for them.

Appearances after Death.—In acute poisoning by the salts of copper, the mucous membrane of the stomach and intestines has been more or less thickened and inflamed in the few fatal cases which have been hitherto examined; the membrane has been also found eroded and softened in poisoning by verdigris. The gullet has presented an inflammatory appearance. In a case of poisoning with verdigris, quoted by Orfila, the stomach was inflamed and thickened, especially towards the pylorus (the intestinal opening), the orifice of which, from the general thickening, was almost obliterated. The small intestines were throughout inflamed, and perforation had taken place, so that part of the green liquid was effused into the abdomen. The large intestines were distended in some parts, and contracted in others, and the rectum was ulcerated on its inner surface. (*Toxicologie*, vol. 1, p. 623.) In some cases the intestines have been found highly inflamed, perforated, and even in a gangrenous state. The lining membrane of the ali-

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ets on the stomach is the ejection of the substance by vomiting. ker assigns the fatal dose at from one to two ounces of verdigris, or e vitriol; but seven drachms have destroyed the life of an adult. quantity of subchloride, equivalent to a scruple, or twenty grains, ved fatal to a child (*ante*, p. 428); and one of the most rapidly fatal s was that of a child, which died in *four hours* from taking an unown quantity of blue vitriol. In *Reg.* v. *Smith* (Monmouth Lent sizes, 1856), prisoner was charged with administering blue vitriol to e prosecutor. It was proved that he had put some crystals of blue triol into a bottle of cider, and the prosecutor suffered from symptoms irritation by reason of his having taken a portion. The fatal dose as here made a subject of inquiry, and the medical witness replied: Half the quantity found in the bottle;" although it is not stated what uantity was found therein. The prisoner was acquitted, on the ground, pparently, that he did not know that bluestone was a "deadly" poi-on. The medicinal dose of sulphate of copper, as a tonic, is from one o three or four grains; and as an emetic, from five to fifteen grains. No other preparation of copper is commonly used as an internal medi-ine in this country.

Treatment.—In general there is violent vomiting, the salts of copper cting powerfully as emetics. The efforts of the stomach should be promoted by the free use of warm water, milk, barley-water, or any mucilaginous drink, and the employment of the stomach-pump. This instrument is of little service when the poison has been taken, as it generally is in coarse powder. Various antidotes have been proposed. Sugar was formerly strongly recommended, on the principle that it had the property of reducing the salts of copper to the state of insoluble and inert red oxide; but this is only under very peculiar circumstances, not likely to be met with in the stomach. Albumen forms an insoluble compound with oxide of copper, provided it is given in large quantity. This and milk may be considered the best remedies.

CHAPTER XLVI.

SALTS OF COPPER—CHEMICAL ANALYSIS—GALVANIC AND OTHER TESTS—DETECTION IN ORGANIC LIQUIDS—PRESENCE OF COPPER IN TRACES—COPPER IN FOOD—ACCIDENTAL POISONING FROM COPPER VESSELS—GERMAN OR NICKEL SILVER—PICKLES AND PRESERVED FRUITS POISONED WITH COPPER.

CHEMICAL ANALYSIS.

THE salts of copper are generally known by their color; whether in the solid state or in solution, they are either blue or green. The salts of one other metal are also of a green color, namely, nickel; but there are striking chemical differences between the salts of this metal and those of copper. There are three *soluble* salts of copper; two of these are blue, the sulphate and nitrate, and one green, the chloride; but this

becomes blue on dilution with water. The salt should be dissolved
water, diluted, and the following tests may be then applied: The
soluble salts may be dissolved in diluted sulphuric or nitric acid,
then tested. The solutions of the cupreous salts generally have an
reaction.

The tests for the oxide of copper are: 1. *Solution of ammonia.* Th
produces a bluish-white precipitate, soluble in an excess of the
forming a deep violet-blue solution. 2. *Ferrocyanide of potassium,*
rich claret-red precipitate. If the quantity of copper be small,
liquid acquires merely a light red-brown color; if large, the preci-
tate is of a deep claret red, and has a gelatinous consistency. The f
rocyanide of potassium will act on the violet-blue solution produced
by ammonia, provided an acid is previously added (sulphuric) to n
tralize the ammonia. One portion of liquid may thus be examined by
the two tests. 3. *Sulphuretted hydrogen gas,* or sulphide of ammonium
gives a deep chocolate-brown precipitate; or, if the copper is in sm
proportion, merely a brown color, either in neutral or acid solution
4. A coil of fine steel wire or a slip of *polished iron* (a common needl
suspended by a thread in the liquid, is speedily coated with a layer
copper, even when the salt is in small proportion. When much dilut
a drop of diluted sulphuric acid may be added, and the iron allow
to remain some hours or days. The iron will be slowly removed,
a hollow cylinder of metallic copper will remain. This may be
solved in diluted nitric acid, and tested with the foregoing tests; or
needle coated with copper may be immersed in a solution of amm
and exposed to air. The liquid then becomes slowly blue, and the
ture of the metal is thus clearly established. Half a grain of sulp
of copper dissolved in sixteen ounces of water, may be thus easily
tested. The presence of organic matter does not interfere with
reaction. It was proposed by Orfila to substitute *phosphorus* for
ished iron. This substance most effectually separates metallic cop
from its salts, but it is not so convenient for use as iron. 5. *The*
vanic test. If a few drops of the copper solution are placed in a
tinum capsule, and slightly acidulated with a diluted acid, and the
tinum is then touched through the solution with a thin slip of
metallic copper, of its well-known red color, is immediately depos
on the platinum. Zinc and platinum wires twisted round each o
may be immersed in the liquid, and allowed to stand some hours, w
the proportion of copper is small. Under these circumstances, t
is merely a reddish-brown stain on the platinum; but a blue liqui
formed by pouring on it ammonia, or it may be dissolved by n
acid, and tested by 1 and 2. By these tests it is easy to detect the 2
part of a grain of a copper salt, or even less. There are no *object*
to the inferences from these tests when taken together; but if reli
be placed on one or two only, the analyst may fall into an error.
action of iron and zinc with platinum, may, however, be considere
conclusive.

The sulphate of copper, used in medicine and chemistry, someti
contains traces of arsenic. About ten grains of the crystallized
phate have yielded evidence of the presence of this poison. When

ate has been given freely as an emetic, traces of arsenic may thus
und in the contents of the stomach or in the matters vomited.
hate of copper is occasionally met with as a fraudulent addition to
l. (Horn's *Vierteljahrsschrift*, 1870, vol. 1, p. 322; also *Med.*
s and Gaz., 1871, vol. 1, p. 509.)

ue vitriol is soluble in four parts of cold and two of boiling water,
is easily obtained in well-defined rhombic crystals by evaporating
all quantity of the solution on a slip of glass. Nitrate of baryta
d to the solution indicates the presence of sulphuric acid.

ere are several varieties of *verdigris*, some of which are blue, and
s green. It is partially soluble in water; but readily when this
dulated with acetic or sulphuric acid. If a portion of the powder
ated in a reduction-tube, a film of metallic copper is produced, and
acid vapor escapes. Acetic acid is, however, readily discovered
oiling the powder in diluted sulphuric acid.

e *insoluble* or partly soluble salts of copper, which may give rise
uestions of poisoning, are the subchloride and carbonate. They
ss these common characters: 1, when rubbed on a steel spatula
a few drops of diluted sulphuric acid, metallic copper is abun-
ly precipitated on the *iron;* and, 2, when treated with a strong
ion of *ammonia*, they acquire a rich violet-blue color. They are
dissolved by diluted nitric acid,—the carbonate with effervescence.

opper in Organic Liquids.—The oxide of copper is liable to be pre-
ated by certain organic principles, *e. g.*, albumen, fibrin, casein, and
ous membrane; but some of these organic compounds are easily
lved by acids, or even by an excess of the solution of cupreous
A portion at least of the salt of copper is, therefore, commonly
dissolved. In such cases there is one peculiar character possessed
hese liquids, *i. e.*, they have a decidedly *green color*, even when the
er salt is in a far less than poisonous proportion, and they have
a strong metallic taste.

polished needle or fine iron wire may be used in these liquids as
al-test for the presence of the salts of copper, or a portion of the
liquid may be placed in a clean platinum capsule, a few drops of
ed sulphuric acid added, and a slip of zinc-foil introduced. Wher-
the platinum is touched by the zinc, metallic copper of its ordi-
red color is deposited; and after having in this way coated the
ior of the platinum capsule with the metal, the surplus liquid may
oured off and the capsule well washed out. The copper may then
issolved in nitric acid, and the tests applied to the solution after
xcess of acid has been driven off by heat. In place of nitric acid
heat a strong solution of ammonia may be used in the cold. Under
sure to air the deposited metal is oxidized and dissolved in a few
ates, forming a blue solution. This ammoniacal solution may then
eutralized with diluted sulphuric acid, and the ferrocyanide of po-
um applied. The red color of the metal, deposited on platinum,
aracteristic of copper; but should any doubt exist, this may be re-
ed by placing a polished needle in the ammoniacal solution and
ng diluted sulphuric acid to neutralize it. The needle is imme-
ely covered with a layer of red copper. Not much importance

mentary canal has throughout presented a deep green color, owing to small particles of verdigris adhering to it. It has been said that this is an uncertain character of poisoning by copper, since a morbid state of the bile often gives a similar color to the mucous membrane of the stomach and duodenum. This objection cannot apply when the green color is found in the gullet, and throughout the intestines; and, under any circumstances, the evidence from the presence of a green color would amount to nothing in the judgment of a prudent witness, unless copper were freely detected in the parts so colored. It is well to remember that the green stains, if due to copper, would be turned blue by ammonia. The liver, stomach, and kidneys have been found congested. In the case of a child poisoned with the subchloride (see *ante*, p. 428), there was nothing to indicate especially the action of an irritant poison, if we except a slight congestion in the cerebral vessels. The child, it appears, had swallowed about a scruple of the green colored substance. It was remarkable that there was not the least sign of irritation or inflammation in the alimentary canal. Death was ascribed to the exhaustion resulting from violent vomiting; and to a congestion of blood in the brain thereby produced.

In the fatal case of poisoning by the carbonate (*ante*, p. 429), the mucous membrane of the gullet and the stomach was covered with the green-colored compound. The larger end of the stomach was reddened and corroded in patches. The mucous membrane of the intestines, as well as the fluid contained in them, was of a green color.

The appearances presented in fatal cases of chronic poisoning by copper are well indicated in one of those which occurred to Dr. Moore (*ante*, p. 429). The mucous membrane of the lower part of the gullet, and that of the stomach between the two orifices, was the seat of extensive and deepseated inflammation. The shades of red varied from a bright vermilion or scarlet to a deep red or violet color. The patches of a dark red or brownish color were comparatively small and circumscribed, situated in general beneath the mucous membrane of the under surface of the stomach. The membrane in these situations was softened, pulpy, but not excoriated, and free from the appearance of having sloughed. At the lesser opening the membrane was intensely inflamed, glistening, and tumid from a quantity of serous fluid deposited beneath the submucous cellular tissue. The mucous membrane of the duodenum and small intestines was also inflamed in irregular patches; and there were traces of inflammation in the large intestines, including the rectum. Eight ounces of a saffron-colored fluid were found in the peritoneal cavity, and on the peritoneal surface of the intestines there were numerous minute spots of inflammatory redness. There was no effusion of lymph or other sign of peritoneal inflammation. (*Lancet*, April 11, 1846, p. 414.)

Fatal Dose.—As the fatal cases of acute poisoning by copper have been but few, it is impossible to assign, with any accuracy, the fatal dose of the salts of this poison. Five drachms of the sulphate have been taken without causing death; and, on the whole, the use of this mineral appears to be more dangerous when taken for some time in small doses, than when a large quantity is swallowed at once. One of the earliest effects on the stomach is the ejection of the substance by vomiting. Böcker assigns the fatal dose at from one to two ounces of verdigris, or blue vitriol; but seven drachms have destroyed the life of an adult. A quantity of subchloride, equivalent to a scruple, or twenty grains, proved fatal to a child (*ante*, p. 428); and one of the most rapidly fatal cases was that of a child, which died in *four hours* from taking an unknown quantity of blue vitriol. In *Reg.* v. *Smith* (Monmouth Lent Assizes, 1856), prisoner was charged with administering blue vitriol to the prosecutor. It was proved that he had put some crystals of blue vitriol into a bottle of cider, and the prosecutor suffered from symptoms of irritation by reason of his having taken a portion. The fatal dose was here made a subject of inquiry, and the medical witness replied: "Half the quantity found in the bottle;" although it is not stated what quantity was found therein. The prisoner was acquitted, on the ground, apparently, that he did not know that bluestone was a "deadly" poison. The medicinal dose of sulphate of copper, as a tonic, is from one to three or four grains; and as an emetic, from five to fifteen grains. No other preparation of copper is commonly used as an internal medicine in this country.

Treatment.—In general there is violent vomiting, the salts of copper acting powerfully as emetics. The efforts of the stomach should be promoted by the free use of warm water, milk, barley-water, or any mucilaginous drink, and the employment of the stomach-pump. This instrument is of little service when the poison has been taken, as it generally is in coarse powder. Various antidotes have been proposed. Sugar was formerly strongly recommended, on the principle that it had the property of reducing the salts of copper to the state of insoluble and inert red oxide; but this is only under very peculiar circumstances, not likely to be met with in the stomach. Albumen forms an insoluble compound with oxide of copper, provided it is given in large quantity. This and milk may be considered the best remedies.

CHAPTER XLVI.

SALTS OF COPPER—CHEMICAL ANALYSIS—GALVANIC AND OTHER TESTS—DETECTION IN ORGANIC LIQUIDS—PRESENCE OF COPPER IN TRACES—COPPER IN FOOD—ACCIDENTAL POISONING FROM COPPER VESSELS—GERMAN OR NICKEL SILVER—PICKLES AND PRESERVED FRUITS POISONED WITH COPPER.

CHEMICAL ANALYSIS.

THE salts of copper are generally known by their color; whether in the solid state or in solution, they are either blue or green. The salts of one other metal are also of a green color, namely, nickel; but there are striking chemical differences between the salts of this metal and those of copper. There are three *soluble* salts of copper; two of these are blue, the sulphate and nitrate, and one green, the chloride; but this becomes blue on dilution with water. The salt should be dissolved in water, diluted, and the following tests may be then applied: The insoluble salts may be dissolved in diluted sulphuric or nitric acid, and then tested. The solutions of the cupreous salts generally have an acid reaction.

The tests for the oxide of copper are: 1. *Solution of ammonia.* This produces a bluish-white precipitate, soluble in an excess of the test, forming a deep violet-blue solution. 2. *Ferrocyanide of potassium*, a rich claret-red precipitate. If the quantity of copper be small, the liquid acquires merely a light red-brown color; if large, the precipitate is of a deep claret red, and has a gelatinous consistency. The ferrocyanide of potassium will act on the violet-blue solution produced by ammonia, provided an acid is previously added (sulphuric) to neutralize the ammonia. One portion of liquid may thus be examined by the two tests. 3. *Sulphuretted hydrogen gas*, or sulphide of ammonium, gives a deep chocolate-brown precipitate; or, if the copper is in small proportion, merely a brown color, either in neutral or acid solutions. 4. A coil of fine steel wire or a slip of *polished iron* (a common needle), suspended by a thread in the liquid, is speedily coated with a layer of copper, even when the salt is in small proportion. When much diluted, a drop of diluted sulphuric acid may be added, and the iron allowed to remain some hours or days. The iron will be slowly removed, and a hollow cylinder of metallic copper will remain. This may be dissolved in diluted nitric acid, and tested with the foregoing tests; or the needle coated with copper may be immersed in a solution of ammonia and exposed to air. The liquid then becomes slowly blue, and the nature of the metal is thus clearly established. Half a grain of sulphate of copper dissolved in sixteen ounces of water, may be thus easily detected. The presence of organic matter does not interfere with this reaction. It was proposed by Orfila to substitute *phosphorus* for polished iron. This substance most effectually separates metallic copper from its salts, but it is not so convenient for use as iron. 5. *The galvanic test.* If a few drops of the copper solution are placed in a platinum capsule, and slightly acidulated with a diluted acid, and the platinum is then touched through the solution with a thin slip of zinc, metallic copper, of its well-known red color, is immediately deposited on the platinum. Zinc and platinum wires twisted round each other may be immersed in the liquid, and allowed to stand some hours, when the proportion of copper is small. Under these circumstances, there is merely a reddish-brown stain on the platinum; but a blue liquid is formed by pouring on it ammonia, or it may be dissolved by nitric acid, and tested by 1 and 2. By these tests it is easy to detect the 250th part of a grain of a copper salt, or even less. There are no *objections* to the inferences from these tests when taken together; but if reliance be placed on one or two only, the analyst may fall into an error. The action of iron and zinc with platinum, may, however, be considered as conclusive.

The sulphate of copper, used in medicine and chemistry, sometimes contains traces of arsenic. About ten grains of the crystallized sulphate have yielded evidence of the presence of this poison. When the sulphate has been given freely as an emetic, traces of arsenic may thus be found in the contents of the stomach or in the matters vomited. Sulphate of copper is occasionally met with as a fraudulent addition to bread. (Horn's *Vierteljahrsschrift*, 1870, vol. 1, p. 322; also *Med. Times and Gaz.*, 1871, vol. 1, p. 509.)

Blue vitriol is soluble in four parts of cold and two of boiling water, and is easily obtained in well-defined rhombic crystals by evaporating a small quantity of the solution on a slip of glass. Nitrate of baryta added to the solution indicates the presence of sulphuric acid.

There are several varieties of *verdigris*, some of which are blue, and others green. It is partially soluble in water; but readily when this is acidulated with acetic or sulphuric acid. If a portion of the powder is heated in a reduction-tube, a film of metallic copper is produced, and acetic acid vapor escapes. Acetic acid is, however, readily discovered by boiling the powder in diluted sulphuric acid.

The *insoluble* or partly soluble salts of copper, which may give rise to questions of poisoning, are the subchloride and carbonate. They possess these common characters: 1, when rubbed on a steel spatula with a few drops of diluted sulphuric acid, metallic copper is abundantly precipitated on the *iron;* and, 2, when treated with a strong solution of *ammonia*, they acquire a rich violet-blue color. They are both dissolved by diluted nitric acid,—the carbonate with effervescence.

Copper in Organic Liquids.—The oxide of copper is liable to be precipitated by certain organic principles, *e. g.*, albumen, fibrin, casein, and mucous membrane; but some of these organic compounds are easily dissolved by acids, or even by an excess of the solution of cupreous salt. A portion at least of the salt of copper is, therefore, commonly held dissolved. In such cases there is one peculiar character possessed by these liquids, *i. e.*, they have a decidedly *green color*, even when the copper salt is in a far less than poisonous proportion, and they have also a strong metallic taste.

A polished needle or fine iron wire may be used in these liquids as a trial-test for the presence of the salts of copper, or a portion of the acid liquid may be placed in a clean platinum capsule, a few drops of diluted sulphuric acid added, and a slip of zinc-foil introduced. Wherever the platinum is touched by the zinc, metallic copper of its ordinary red color is deposited; and after having in this way coated the interior of the platinum capsule with the metal, the surplus liquid may be poured off and the capsule well washed out. The copper may then be dissolved in nitric acid, and the tests applied to the solution after the excess of acid has been driven off by heat. In place of nitric acid and heat a strong solution of ammonia may be used in the cold. Under exposure to air the deposited metal is oxidized and dissolved in a few minutes, forming a blue solution. This ammoniacal solution may then be neutralized with diluted sulphuric acid, and the ferrocyanide of potassium applied. The red color of the metal, deposited on platinum, is characteristic of copper; but should any doubt exist, this may be removed by placing a polished needle in the ammoniacal solution and adding diluted sulphuric acid to neutralize it. The needle is immediately covered with a layer of red copper. Not much importance should be attached to the discovery of mere traces of this metal either in the body or in articles of food. Copper, if looked for, may be found in many cases in the tissues and in food, under circumstances quite unconnected with poisoning. It was found in a mutton chop procured fresh from a butcher's shop.

Assuming that either of these trial-tests indicates the presence of a cupreous salt in a large proportion, we may boil the liquid, and destroy its viscidity by diluting it, if necessary, and, after filtration, we may pass into it a current of sulphuretted hydrogen gas in order to precipitate the copper in the state of sulphide. The black sulphide may be collected, washed, dried, and then boiled in equal parts of nitric acid and water for a quarter of an hour. Nitrate and sulphate of copper are produced and dissolved—a fact indicated by the liquid acquiring a rich blue color; and some sulphur is at the same time separated. This liquid, when filtered and diluted, will give the usual reactions with the tests for copper.

The sulphide of copper thus obtained, when washed and dried, may serve for determining the quantity of cupreous salt present. For this purpose a portion of it should be transformed into black oxide of copper by digesting the sulphide in strong nitric acid, evaporating the acid, and incinerating the residue.

Mucous and other organic viscid liquids may be placed in a dialyzing vessel and treated like arsenic and mercury (*ante*, p. 154). A soluble copper salt, if present, is readily obtained by *dialysis* in a state fitted for the direct application of the tests as already described for the sulphate.

For the *tissues*, *urine*, and solid *articles of food*, which do not show the presence of copper by dialysis and the usual tests, one process only is applicable, *i. e.*, incineration. The substance must be first brought to a perfectly dry state and then completely burnt to an ash in a platinum crucible. We may digest the residuary ash in pure hydrochloric acid by heat, and then evaporate nearly to dryness. The residue may be dissolved in a small quantity of water, and a polished needle immersed for some hours. The metallic deposit, if any, on the needle, may be recognized as copper, either by its color or by the action of ammonia.

Copper is often found in traces by incineration in the tissues of the body and in articles of food. No importance can be attached to this discovery unless there have been symptoms of poisoning produced. Some have described copper as a *normal* constituent of the body, but there is reason to believe that when found, it has been accidentally received from without through food, and that it is really a portion of the metal which has not undergone elimination. See paper by Drs. Odling and Dupré. (*Guy's Hosp. Rep.*, 1858, p. 104.)

As an objection to medical evidence of poisoning, the admission of its occasional presence in the body, or in common articles of food, has no force: 1. Because in poisoning by copper there would be very few cases in which the whole of the chemical evidence rested on traces of the metal found by an incineration of the viscera. Such a case is very unlikely to occur; for chemical evidence is in general abundantly afforded by an analysis of a portion of the poisoned substance swallowed, or of the contents of the stomach. 2. If the only chemical evidence were that derived from incineration, then this could afford no proof of poisoning, unless that fact were already sufficiently made out by symptoms, appearances, and moral circumstances, in which case such infinitesimal proof might be easily dispensed with. In a case of falsely imputed poisoning, it may be said that the detection of copper in a particular article of food, such as bread, would lead a medical jurist into error, since the discovery of this metal in the bread might bear out the imputation, and inculpate an innocent person. This hypothesis does not appear probable. The normal copper, said to exist in food, has not been found to form, according to its discoverers, more than the 100,000th part of the food examined; if the imputation of poisoning were well-founded, and copper were discovered at all, the metal would be in infinitely larger proportion than this, so as to leave no doubt of its actual admixture. These facts furnish an objection, therefore, only to the evidence of those who rely exclusively on the infinitesimal results of a chemical analysis.

A herbalist was recently tried in France for poisoning two wives with sulphate of copper. The first wife suffered from cramp in the stomach with glairy mucous vomitings; she vomited nearly all her food, and suffered violent pain at the pit of the stomach. The body was not examined until seven months after death, and the viscera were found in a good state of preservation. The second wife, according to another medical witness, suffered from incessant vomiting, pains in her limbs, and failure of eyesight, and she died from exhaustion. No natural cause of death was detected. The symptoms were ascribed to sulphate of copper. They were similar in the two cases. On analysis, copper was detected in the liver and kidneys, but not in the stomach or intestines. From a fourth part of the organs removed from the bodies of the two women, the chemist separated 30 milligrammes (= 0.46 grain) less than half a grain of the metal. They treated this as a positive proof of poisoning by copper, and the man was convicted of murder. (*Brit. Med. Jour.*, Sept. 1874, p. 407.) The medical witnesses denied that this could have been normal or accidental copper, on the ground that the quantity found by them was too large. It seems they examined the livers of fourteen dead subjects, and they found only one milligramme ($\frac{1}{65}$th of a grain) of copper. One of the samples of earth collected from the graves of the deceased women, also contained traces of copper. It is not improbable that the two women died from the effects of a copper salt, producing exhaustion by incessant vomitings, and, that the circumstantial and general evidence was sufficient to justify the verdict; but the chemical evidence failed to show satisfactorily that sulphate of copper had been administered. The quantity found was so small as to be consistent with an accidental introduction of the metal in food.

It has been suggested that one source of error might exist as the result of the use of gas jets containing copper, a portion of the metal being carried over during incineration with the carbonaceous residue of the organic matter. It is not easy to perceive how any properly conducted analysis could be so affected as to lead to error from this source. It furnishes an argument against an undue reliance upon traces of metal.

It is not probable that a medical jurist would be required to seek for a cupreous poison in a body which had been so long interred that the remains were intermixed with the soil. But it is not the less necessary to state that, according to the researches of M. Walchner, copper, like arsenic, is almost universally found in ferruginous soils, and in most kinds of marls and clays. Wherever the ores of iron exist, there copper will be found; in this way it may be dissolved in water, and percolate through the superficial strata. (See *Comptes Rendus*, Sept. 21, 1846, p. 612.) Admitting the truth of this observation, a comparative analysis of the earth of the cemetery would be required in the very rare case in which the decomposed remains of the dead had become intermixed with the soil. M. Walchner simply digested the earth in muriatic acid, and precipitated the copper from the acid solution by a current of sulphuretted hydrogen gas.

Copper in Food.—The medico-legal history of poisoning by copper would be incomplete without some remarks on the action of certain articles of food on this metal, when it is used for culinary purposes. This is not an unfrequent form of accidental poisoning. The symptoms rarely appear until after the lapse of three or four hours, or even a much longer period. There is commonly nausea, with colicky pains and cramps in the limbs. It results, from the experiments of Falconer and others, that metallic copper undergoes no change by contact with *water*, unless the air is present, when a hydrated carbonate mixed with oxide of copper is formed. If the water contains an acid, such as vinegar or common salt, or if there is oily or fatty matter in contact with the metal, then the copper is more rapidly oxidized, and the liquor or fat acquires a green color. If the copper vessel is kept perfectly clean, and the food prepared in it is allowed to cool in other vessels, there is not much risk of its acquiring a poisonous impregnation; nevertheless, no acid, saline, fatty, or oily liquid should be prepared as an article of food in a copper vessel. (See *Ann. d'Hyg.*, 1832, vol. 1, p. 102.) Under the influence of heat and air a portion of copper becomes dissolved, and the oily or other liquid acquires a green color. The preparation of fruits, such as preserves, in copper vessels, is necessarily attended with some risk; for, on cooling, a green crust is apt to be formed on the copper just above the surface where the air and acid liquid meet. Some liquids while boiling are but little liable to this impregnation; thus coffee, beer, milk, and tea have been separately boiled for two hours together, in a clean copper vessel, without any portion of the metal being taken up by either of the liquids. (See Falconer, *On the Poison of Copper*, p. 65, London, 1774; also *Orfila*, vol. 1, p. 611.) Accidents of this kind are usually prevented by lining the copper vessel with tin; but in very large boilers this plan is not always adopted, cleanliness alone is trusted to, and this when properly observed is a sufficient preventive. In reference to culinary vessels the tin is often worn away, and the corroded copper is thus exposed to the action of any acids contained in the food. Mr. Todd, coroner for Hants, communicated to me the following case (Aug. 1866): Some rhubarb-stems were stewed in a copper vessel imperfectly tinned and dirty, and were supplied to a family for dinner. The children and their governess partook of the food, the latter very freely. All were taken ill. The governess suffered most; there was violent sickness, with other symptoms of irritation. She recovered partly under treatment, but had a relapse and died from the effects of the poisoned food. The oxalic and malic acids in the vegetables probably acted strongly on the copper.

In July, 1866, a remarkable set of cases occurred in the family of a Mr. Corrie, Itchen Abbas, Hants, in which twelve or more members of the family suffered from symptoms of poisoning similar to those produced by copper in food. A badly tinned copper vessel had been used for cooking the food, with much salt. One patient, an old man, who partook of the food, æt. 90, died after three weeks, the others recovered. The cook was charged with wilful poisoning, but was subsequently liberated. She brought an action against her master (*Tully* v. *Corrie*, Queen's Bench, Nov. 1867), but this resulted in a verdict for the defendant. A full account of this case will be found in the *Guy's Hosp. Rep.*, 1866, p. 329. A set of cases is reported to have occurred at Geneva in 1870, in which ten persons were taken ill with symptoms of irritant poisoning, and four died. It was found that the food had been cooked in a copper vessel containing a large quantity of verdigris. (*Phar. Jour.*, Aug. 1870, p. 158.) A fatal case of poisoning by copper is reported in the same journal for 1870, p. 874. Copper was found in small quantity in the tissues of the body. Dr. Waldemann, of Erfurt, has lately published an elaborate paper on the effects of copper and zinc and their alloy, brass, when used for culinary utensils. (Horn's *Vierteljahrsschrift*, 1870, vol. 1, p. 247.)

The tin used for lining copper vessels is frequently alloyed with a large proportion of lead, and thus lead-poisoning may be substituted for poisoning with copper. According to Paasch, of Berlin, many of the accidents attributed to this form of cupreous poisoning are really due to other causes. (Casper's *Vierteljahrsschrift*, 1852, vol. 1, p. 78.) It has been elsewhere stated that all the ordinary copper employed for culinary utensils, contains arsenic. In those cases in which the metal is converted into insoluble oxides or salts by acids or fat, the arsenic is found in an insoluble form in the green incrustation produced. When copper thus forms an insoluble salt, I have not found any arsenic in a dissolved state.

Accidental poisoning by copper has occurred from the use of what is called *German silver*, but which should rather be called *white brass*, as it is an alloy of copper and zinc with nickel. Some specimens of this alloy contain fifty per cent. by weight of copper. The following case of poisoning occurred in Paris in 1838: A lady, after having had eels for dinner, was awakened in the night by intense headache, followed by nausea, vomiting, and severe colic. These symptoms were removed under proper treatment. Her physician ascertained that the eels had been cooked with butter and vinegar in an earthenware vessel, and he found that the metal spoon, which was of German silver, presented on different parts greenish-colored spots. Chemical analysis showed that a poisonous salt of copper had been thus accidentally produced—a fact demonstrated by polishing the spoon and then placing it in a hot mixture of bread, butter, and vinegar. Half an hour after the mixture had cooled, green spots were perceived on it, and in twelve hours the spoon was quite green as well as the butter in contact with it.

It has been stated that an impure gold alloy used by some of the lower class of dentists has been so largely composed of copper as to affect the health of those who have used the plates for the support of artificial teeth. The acid and salts in the saliva facilitate the production of a poisonous salt of copper, and probably set free arsenic.

In the making of preserved *fruits* and vegetable *pickles*, the salts of copper (blue vitriol) are sometimes used for the purpose of giving a rich green color! Many of the green pickles sold in shops are thus impregnated with the vegetable salts of this metal, to which they owe their bright grass-green color. If the fruit or pickle is placed in a solution of ammonia, and copper is contained in it, the substance is speedily turned blue. The iron-test is, however, more delicate. A bright needle immersed in the pickle, or plunged into the solid, will be speedily coated with copper. The quantity of copper contained in such articles may not be sufficient to cause fatal effects; but serious symptoms of gastric irritation are sometimes produced, and in young persons these may assume an alarming character. (See *Falconer*, p. 87.)

On one occasion some preserved gooseberries were sent to me for examination, as it was suspected, from their having produced symptoms of poisoning in a child, that they were contaminated with copper. The suspicion turned out to be correct. The cook had mixed with them some blue vitriol to improve the green color. Dr. Hassall states that he found copper in sixteen different samples of London pickles, and it was most abundant in those which were green. (*Food and its Adulterations*, p. 388.)

Some proceedings taken under the Adulteration of Food Act (July, 1874) have shown that this noxious practice still continues. A dealer was convicted of selling green peas which owed at least a part of their color to sulphate of copper. They were prepared in France for sale in England!

A few years since a fraudulent practice existed on the continent of mixing sulphate of copper with the dough of bread. The quantity of cupreous salt used was small, but still it was a noxious adulteration. (*Ann. d'Hyg.*, 1830, p. 342; 1831, p. 338; 1840, vol. 2, p. 123.) According to some experimentalists, bread always contains traces of copper, which may be derived from the blue vitriol with which seed corn is frequently dressed. (*Annuaire de Chimie*, 1846, p. 686.) It may also have been introduced accidentally during the making of the bread, as where copper utensils have been used for this purpose. Thus it may be found in bread, and not in the flour from which the bread is made, or in the flour and not in the corn. M.M. Theulen and Servan having found copper in a specimen of bread, ascertained by further examination that copper cylinders have been used in grinding the corn. A small quantity of oxide falling from these would at once account for the contamination irrespective of fraud. (See *Orfila*, vol. 1, p. 651; *Galtier*, vol. 1, p. 607.)

CHAPTER XLVII.

POISONING WITH ANTIMONY—TARTAR EMETIC—SYMPTOMS—ACUTE AND CHRONIC POISONING—EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE, AND PERIOD OF DEATH—TREATMENT—DETECTION OF THE METAL IN ORGANIC LIQUIDS AND SOLIDS.

ANTIMONY.

General Remarks.—METALLIC ANTIMONY is not regarded as a poison, but when respired in the state of *vapor*, it is stated to have produced serious symptoms. A case of poisoning by the vapors of antimony is reported in the *Edinburgh Medical and Surgical Journal* (vol. 5, p. 265). Orfila suggests that the effects said to have been produced by this metal in vapor, may be ascribed to arsenic, which is present in most specimens of crude antimony as it is used in manufactures. (*Toxicol.*, vol. 1, p. 504.) Of the antimonial compounds, there are only two which require special consideration, namely *Tartar emetic* and *Chloride of antimony.*

TARTARATED ANTIMONY—TARTAR EMETIC—STIBIATED TARTAR.

Tartar emetic owes its poisonous properties to the oxide of antimony, of which it contains 44 per cent., the residue being composed of potash, tartaric acid, and water. In consequence of its having been frequently given to adults in large doses without causing death, its poisonous properties have been doubted. This subject has already been fully considered (*ante*, p. 70). One reason why the symptoms are often so slight from comparatively large doses, is owing to its possessing such violent emetic properties. This leads to the early expulsion of the greater part of the poison from the stomach. When given in small doses at intervals the effects are those of chronic poisoning. Common medicinal doses often produce much vomiting and great depression. A case is related by Dr. Lambert, in which only four grains of tartar emetic gave rise to violent pain in the abdomen, vomiting, and purging.

It appears, from the observations of the late Mr. Goodlad, of Manchester, and Mr. Noble, that tartar emetic, even in small doses, is liable to act as a poison on the young. Mr. Wilton records four cases in which prostration and collapse followed the administration of ordinary doses of tartar emetic to young children. Two of them were fatal. Three-quarters of a grain of tartar emetic were prescribed for an infant recovering from measles. The child died in an hour from the depressing effects of the medicine. A similar dose was prescribed for another child of the same parents: violent vomiting and purging supervened, and this case also ended fatally. In a third instance of a girl, æt. 4, suffering from hooping-cough, one-third of a grain given in divided doses produced alarming symptoms, which rendered a discontinuance of the medicine necessary. (*Journal de Chimie*, Sept. 1847, p. 471; see also *Med. Gaz.*, vol. 40, p. 351.)

Tartar emetic acts more as an irritant than as a corrosive; but the symptoms which it produces, like those of all corrosive poisons, are generally immediate—some, at least, are manifested within a few minutes. It is used in medicine both externally and internally. VINUM ANTIMONIALE is a solution of tartar emetic in sherry wine; it contains two grains in an ounce. It is prescribed in doses of ten drops to one drachm, and in much larger doses as an emetic. It has no taste. Tartar emetic ointment contains one-fifth of its weight of this substance.

Symptoms—Acute Poisoning.—When tartar emetic is taken in a dose of from one to two or three drachms or upwards, the person experiences a strong metallic taste, which continues for some time. In a few minutes there is nausea followed by incessant vomiting, which continues generally until the stomach is cleared, and even for some time afterwards, as a result of local irritation. There is pain in the stomach and bowels, followed by purging, more or less violent; a sense of burning heat and constriction or choking in the throat, extending through the whole length of the gullet to the stomach, difficulty of swallowing, soreness of the mouth and throat, followed by the peeling off of the lining membrane, or the formation of an aphthous crust, at first whitish, but becoming subsequently discolored, brown and black. When this symptom occurs, it is probable that the same condition of the mucous membrane exists in the gullet, stomach, and part of the intestinal canal. In some cases there is great thirst, with increased flow of saliva. The vomited matters consist of a white stringy mucus, locking up solid portions of the poison, but sometimes tinged with blood or bile; the evacuations are liquid and bilious. There are cramps in the arms and legs; sometimes there are severe tetanic spasms; coldness of the surface, with clammy perspiration, attended with flushing, and a congested state of the head and face, faintness, and a feeling of extreme depression, loss of muscular power, pulse small, contracted, and feeble—in advanced cases fluttering, and barely perceptible; respiration short and painful, livid or dusky appearance of the lips and face, especially around the eyes, which are sunk; loss of voice, complete incapacity for any exertion; an eruption resembling that of small pox occasionally showing itself on the skin; wandering or delirium, with loss of consciousness.

These symptoms are not met with in every case; thus, vomiting and purging may coexist, or one may be vicarious of the other. In certain cases, neither of these symptoms may be present, and then those affecting the nervous system are generally more prominent. The intensity of the symptoms, the rapidity of their progress, and the speedy access of collapse, chiefly distinguish those of the *acute* from the *chronic* form. In the latter variety there is nausea, a loathing of food and incessant retching, without actual vomiting until food is taken. The vomited matters are sometimes white (mucus), but at a later period covered with bile, and the symptoms recur with severity after each administration of the antimony in food or medicine, the prostration of strength being great in proportion to the frequency of this recurrence.

There has been noticed a greatly increased secretion of urine. In no instance has suppression been observed, as in cases of arsenical poisoning. Antimony appears to be carried off abundantly by the urine. In the acute form of poisoning, the presence of poison in the food may generally be perceived by the taste; in the chronic form, from the smallness of the quantity, there may be no taste perceptible.

One of the remarkable characters of the acute form is that, in spite of the violence and severity of the symptoms, even when the collapse and depression appear to indicate impending dissolution, there is an astonishing power of recovery. When one large dose only is administered, the case proceeds steadily to recovery or death, generally the former if the case is placed early under proper treatment. In this respect acute antimonial is distinguished from acute arsenical poisoning. In the latter, in spite of early treatment, and the removal of the whole or the greater part of the poison from the stomach, the case frequently terminates fatally. Should, however, another dose of antimony be taken at or about the time at which recovery is taking place from the effects of the first, it will be easily understood that the person will sink under the effects of the poison. If any doubt exist concerning the cause of the symptoms, *i. e.*, whether they be due to bilious cholera or some form of gastritis or gastro-enteritis, then an examination of the urine should be made. If this be examined at intervals, it will be found to contain antimony, should the case be one of antimonial poisoning. An analysis directed to the matters vomited and the excreta, will also aid the practitioner in forming an opinion.

In a case reported by Mr. Freer, a man æt. 28, swallowed half an ounce (240 grains) of tartar emetic by mistake for Epsom salts, and recovered from its effects. An hour after the poison had been taken, he was found in the following state: His pulse imperceptible; tongue dry and red; countenance cold and livid, bathed with clammy perspiration, and indicative of great suffering; violent pain in the stomach, and over the whole of the abdomen, with constant spasmodic contraction of all the muscles, particularly of the abdomen and arms. The fingers were firmly contracted, and the muscles quite rigid. He vomited only once, about *half an hour* after he had swallowed the poison, and after this he had constant involuntary aqueous purging. An emetic of mustard and salt was given to him, and this produced violent vomiting of bilious matter. Green tea, brandy, and decoction of oak-bark, were freely given. The cramps, vomitings, and aqueous purging continued for six hours. The symptoms then became mitigated, and he gradually recovered, suffering chiefly from profuse night perspirations. (*Lancet*, May 22, 1847, p. 535.) This case is remarkable for the anomalous character of the symptoms, as in the absence of active vomiting, an emetic was actually required to be given; also for the recovery of the individual after a very large dose of the poison.

I am indebted to Mr. Couling, of Brighton, a former pupil, for a case of recovery from a large dose, which occurred in his practice in July, 1866. A veterinary surgeon swallowed by mistake for carbonate of soda about 200 grains of tartar emetic in powder. He noticed a peculiar taste. Vomiting came on in fifteen minutes, but only after tickling his throat. This continued violently. In two hours there was severe purging with symptoms of collapse. The vomited matters were green, and the evacuations like boiled sago. There was no appearance of blood in either. In three hours severe cramps came on, affecting all the muscles; he was unable to move or speak. Brandy and other remedies were employed, and in six hours, after a warm perspiration, he began to recover. There was suppression of urine. Only a small quantity was passed, and this was of a coffee color. For two or three days he suffered from stiffness in the limbs and in the muscles of the abdomen. In one instance a small dose of this substance caused death by producing intestimal hemorrhage. (*Assoc. Med. Jour.*, June 10, 1853, p. 513.) Mr. Procter, of York, communicated to me, in July, 1860, the cases of four children, to whom, by mistake, a mixture of sulphur and tartar emetic had been given. An ounce of sublimed sulphur and one drachm of tartar emetic had been divided among the four. The symptoms presented the same characters in each; early vomiting, which became violent and incessant, pain in the bowels, purging, great thirst, cold clammy perspiration, feeble pulse, cramps of the limbs and twitchings of the muscles with great depression. There was no sense of heat or constriction in the throat, and no difficulty of swallowing. Under treatment they all recovered.

Dr. Gleaves, U. S., has related, in the *Western Journal of Medicine and Surgery*, the following case: A young man swallowed by mistake a *tablespoonful* of tartar emetic (= about 478 grains). In an hour afterwards he was speechless, pulseless, and apparently dying. Although he drank freely of cold water, and irritated his throat repeatedly with his finger, no vomiting had occurred. During the first three hours he vomited only two or three times, and the matter ejected was chiefly the water taken to favor vomiting. After the lapse of two hours there was violent purging. In seven hours this ceased, and there was great thirst, with a sense of burning pain in the throat, gullet, stomach, and bowels. There was also great irritability of the stomach, and the vomited matters were tinged with blood. On the following day the vomiting continued, but the purging was arrested. The throat was covered with pustules; there was pain in passing the urine, which was copious. On the third day, the whole of the body was covered with genuine tartar emetic pustules. These began to heal, and the patient to recover, in about two weeks. (*Medical Times*, Jan. 24, 1846, p. 127.) This is the only case of poisoning with tartarized antimony, in which pustular eruptions on the skin are stated to have been observed. It is otherwise remarkable for recovery from so large a dose, considering that but little of the poison could have been expelled in the first instance by vomiting.

Chronic Poisoning.—A good account of the effects produced by this poison, given at intervals in small doses to healthy persons, has been published by Dr. Mayerhofer. (Heller's *Archiv.*, 1846, pts. 2, 3, 4, p. 100, *et seq.*) The principal symptoms observed were—great nausea, vomiting of mucous and bilious liquids, great depression, watery purging, followed often by constipation of the bowels; small, contracted, and frequent pulse; loss of voice and muscular strength; coldness of the skin, with clammy perspiration, and death from complete exhaustion. Several cases have recently occurred in this country, which show that tartar emetic has been thus criminally and fatally used. In addition to the cases of Ann Palmer and J. P. Cook, there are those of *Reg.* v. *McMullen* (Liverpool Summer Assizes, 1856), *Reg.* v. *Freeman* (Drogheda Spring Assizes, 1857), and *Reg.* v. *Hardman* (Lancaster Summer Assizes, 1857), the cases of the James family at Liverpool, *Reg.* v. *Winslow* (Liverpool Autumn Assizes, 1860, *ante*, p. 121), *Guy's Hosp. Rep.*, Oct. 1857, and the case of General Ketchum (*ante*, p. 111).

External Application.—Tartar emetic is said to have produced symptoms of irritant poisoning when applied externally to the skin in the form of ointment as a counter-irritant. In a case where the skin was but little affected by the use of this ointment, nausea and sickness were produced, which disappeared when the use of the ointment was discontinued. Although it is extensively used as an external application by medical practitioners, it is rare to hear of cases of poisoning by it under these circumstances. Dr. Griffiths, of Philadelphia, states that, among other symptoms, it has produced violent salivation. The effects appear to be usually limited to the production of local irritation and a pustular eruption on the parts of the skin to which it has been applied.

Appearances after Death.—The mouth, throat, and gullet have been found inflamed, or in an aphthous state. The inflammation has been sometimes confined to the mucous membrane of the throat and the lower part of the gullet. The mucous membrane of the stomach is more or less reddened in patches or spots, as a result of inflammation; the membrane is softened or corroded, and easily removed by friction, sometimes covered with false membrane or aphthous crusts; the surface darkened, inflamed, and ulcerated; and small ulcers with pustular exudations, are occasionally found. The contents of the stomach are of a dark brownish color, consisting chiefly of mucous matters, colored either by blood, bile, or by a mixture of both. The peritoneal or external coat of the stomach has been found inflamed; the intestines present similar appearances, the inflamed portions of mucous membrane being seen chiefly in the duodenum, cæcum, and rectum; the contents of the intestines are bilious or bloody, with much mucus. There are aphthous ulcerations in the glands of the small intestines; the lungs show more or less congestion in portions of the lobes; the heart is empty, or if blood be contained in its cavities this is dark-colored and liquid; the blood liquid throughout the body. The brain and its membranes have been found congested, and the substance of the brain softened. Cases have been met with in which these appearances have not been found, or the changes have been slight and unimportant. (Böcker, *Vergiftungen*, p. 37.) In death from *chronic* poisoning, the liver has been found enlarged, and so softened, that its structure was easily broken down. The organs of the body have been in some instances well preserved. These appearances will necessarily vary according to the duration of the case. When life is protracted, there may be the appearances of gastro-enteritis in a severe form.

Two children, a boy æt. 5 years, and a girl æt. 3 years, each swallowed a powder containing *ten grains* of tartarized antimony mixed with a little sugar. It was stated that, in twenty minues after taking the powders, they were seized with violent vomiting and purging, and great prostration of strength, followed by convulsions and tetanic spasms; there was also great thirst. The boy died in eight hours, and the girl in twelve or thirteen hours, after swallowing the dose. The bodies were inspected between four and five days after death. In that of the boy there was effusion of serum in the right pleura; the lower lobe of the right lung posteriorly was redder than natural, and the peritoneum was injected from recent inflammation. The mucous membrane of the duodenum was inflamed, and covered with a whitish-yellow viscid secretion; this was observed throughout the intestinal canal, although the color was of a deeper yellow in the large intestines; there was no ulceration. The peritoneal coat of the stomach was inflamed. The mucous membrane of this organ was much inflamed, especially about the larger curvature, and at the cardiac orifice; there was no ulceration. The contents (about two ounces and a half of a dark grumous fluid, having a slightly acid reaction) were adherent to it; and in one case there was a patch of lymph. The tests used did not indicate the presence of antimony. With regard to other appearances, the tongue was covered with a white fur, and appeared soddened; the fauces were not inflamed; the windpipe and gullet had a natural appearance. On opening the cranium, the dura mater was found congested; the longitudinal sinus contained a coagulum of lymph, and but little blood. The vessels of the surface of the brain were much injected with dark blood, the whole surface having a deep purple color. Every portion of the brain, when cut, presented many bloody points. The cerebellum and medulla oblongata were also congested; there was no effusion in the ventricles, or at the base of the brain. In the body of the girl, the morbid appearances were similar; there were also patches, resembling the eruption of scarlatina, on the arms, legs, and neck. The arachnoid membrane was more opaque than usual; and on the mucous membrane of the stomach, where the inflammation was greatest, were two or three white spots, each about the size of a split pea, which appeared to be the commencement of ulceration. (Mr. Hartley in *Lancet*, April 25, 1846, p. 460.)

A girl, æt. 16, swallowed a dose of tartarized antimony, amounting to from forty to sixty grains. There was severe vomiting in a quarter of an hour, and this was soon followed by purging; these symptoms continued for about three hours. She also complained of pain and a burning sensation down the œsophagus. The vomited matters were of a dark color. On the following morning she had recovered from the severity of the symptoms; but in the afternoon there was a relapse. She continually threw her head back, and screamed; the skin was warm and moist; the pupils were dilated; and the knees drawn up. She died in about thirty-six hours after taking the poison, and during the six or eight hours previous to her death she was quite delirious. An inspection was made thirty-six hours after death. The throat appeared swollen; the lungs were slightly congested; the heart was healthy, and contained about six drachms of fluid blood. The stomach contained sixteen ounces of a thick bloody liquid; at the greater extremity the coats were softened, and blood was effused under the mucous coat in several places. The small intestines contained a similar liquid with much mucus; but there was no appearance of inflammation. Only slight traces of the poison were found in the contents of the stomach by the usual tests, the greater part having probably passed off by vomiting and purging. (Mr. Beale in *Lancet*, Jan. 21, 1854.) In animals poisoned by this substance, Dr. Pavy and I have found general inflammation of the lower half of the alimentary canal.

Fatal Dose—Period of Death.—The *quantity* of tartar emetic which is actually required to destroy life is unknown. It will probably depend in a great degree on whether active vomiting and purging have been excited or not; for these symptoms have not been present in all cases. Doses of from twenty grains to one ounce have been taken without destroying life; although alarming symptoms of irritation have followed. In one case related by Orfila, a man, æt. 50, took forty grains of tartarized antimony, and died in about four days. This was the only one out of about five cases of poisoning by this substance quoted by Orfila, which proved fatal. (*Orfila*, vol. 1, p. 480.) Dr. Beck mentions a case in which fifteen grains of this substance in solution killed a child in a few weeks; vomiting and purging ensued, followed by convulsions and death. This case proves that a patient is not always saved by vomiting and purging; the fatal effects on such an occasion are probably due to rapid absorption. (See also *Medical Gazette*, vol. 44, p. 334.) Dr. Pollock has recorded a case in which an adult was killed in ten hours by a dose of one drachm, in spite of early and violent vomiting. (*Med. Gaz.*, vol. 45, p. 801.) In the two cases observed by Mr. Hartley (p. 446) a dose of *ten grains* proved fatal to each child in a few hours. A dose of four grains, however, has been known to produce alarming symptoms. Dr. Lambert, who reports the case in Casper's *Wochenschrift* (1841), states that this dose gave rise to violent pain in the abdomen, vomiting, and purging. The patient then fell into strong convulsions, which lasted half an hour. He became speechless, no pulse could be perceived, the skin was cold, and it was supposed that he was dead. Stimulating frictions and poultices were employed, and he slowly recovered in about fourteen days.

A case was referred to me in March, 1847, in which it was of some importance to assign the probable fatal dose for a child æt. 4 or 5. The child was laboring under disease of the lungs. An antimonial mixture was prescribed for it. Two doses were taken, and the child died twenty-four hours after taking the last dose. The cause of death was assigned to the medicine, and the medical attendant was charged with manslaughter. The child had not suffered from vomiting or purging, or other symptom, excepting pain after taking the medicine. I found by examining the antimonial mixture, that each dose contained a quarter of a grain of tartar emetic, making half a grain in the whole. From this result, and from the absence of the usual symptoms of antimonial poisoning, an opinion was given that the child had not died from the effects of the medicine, but from the disease.

Tartar emetic in small doses may occasion death by reason of its exerting a depressing influence on the action of the heart. Aged persons, or those who are debilitated by disease, might die under these circumstances from a dose or doses which would produce no injury to strong and healthy adults. The effects, however, should be clearly traced to the action of the poison, and not be owing to exhaustion as a result of disease. In February, 1853, Mr. Wakley referred to me for examination a case, in which it was supposed that two doses of antimonial wine, equal to about *three grains* of tartar emetic, had caused the death of a man who was in a diseased condition, by its remote effect upon the heart. No trace of antimony was found in the stomach or tissues, there were no symptoms to indicate poisoning, and under these circumstances death could not be reasonably attributed to the medicine. The man died in about twenty hours afterwards, probably from exhaustion of the vital powers as a result of disease, and not from the direct action of the substance.

It has been generally supposed that the cases in which this poison has proved fatal have been but few; but I have elsewhere reported thirty-seven, of which sixteen were fatal. The smallest fatal dose was in a child,—*three-quarters of a grain*, and in an adult, two grains; but in this case there were circumstances which favored the fatal operation of the poison. (*Guy's Hospital Reports*, Oct. 1857.)

The medicinal dose of tartar emetic, in substance, as a sudorific and expectorant, is from one-twelfth to one-eighth of a grain; to produce nausea, one-quarter to one-half grain; to act as an emetic, one to two grains. In the treatment of inflammatory diseases of the lungs it has been used in much larger doses, although not without dangerous result. (See p. 70.)

Taking the facts hitherto collected, it appears probable that under circumstances favorable to its noxious operation on the system (indicated by failure of pulsation and collapse), a dose of from *ten* to *twenty grains*, taken at once, might destroy an adult, and if taken in divided doses, a smaller quantity than this might suffice. Large doses are very uncertain in their operation. In two instances persons have recovered after taking quantities twice, and even eight times, as great as that which has proved fatal to a healthy man.

In reference to the fatal dose, it is rather to the effects produced than to the actual quantity swallowed, that we are to look. As vomiting and purging generally occur speedily, and the poison is known to be ejected, it would be irrational to assume that the dose swallowed remained unaltered in the body. It is important, therefore, not to fix the fatal dose by actual weight. A quantity which may destroy an infant will not destroy an adult woman, and a dose which may kill a delicate woman, or an old person, might not act fatally on a strong and healthy man. A person laboring under disease may be more easily destroyed than one who is healthy, and lastly, there is that ever-varying condition of idiosyncrasy, in which, as it is well known, there is a state of constitution that renders a person more liable to be affected by antimonial compounds than other persons apparently in precisely the same conditions as to health, age, etc. Then, again, a dose of ten grains, administered at once, may not be attended with the same amount of danger to life as the same quantity given in small doses over many days or weeks.

Treatment.—This consists in promoting vomiting by the free administration of tepid water, with milk, or other diluents. A solution of tannic acid may be given at short intervals or injected by the stomach-pump. In the absence of tannic acid any vegetable infusion containing it, such as strong green tea, decoction of oak-bark, or Peruvian bark, may be given. This principle combines with oxide of antimony to form a compound insoluble in water; and, if attended with no other benefit, it at least suspends the operation of the poison.

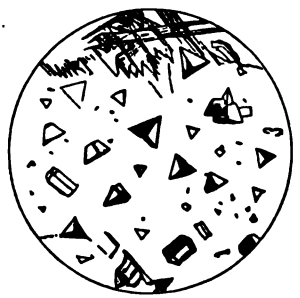
CHAPTER XLVIII.

CHEMICAL ANALYSIS—TARTAR EMETIC AS A SOLID AND IN SOLUTION—DETECTION OF ANTIMONY IN ORGANIC LIQUIDS AND SOLIDS—QUANTITATIVE ANALYSIS—DETECTION IN THE TISSUES AFTER LONG PERIODS.

CHEMICAL ANALYSIS.

Tartar Emetic as a Solid.—In the state of powder it is white and crystalline. It has been occasionally sold by mistake for tartaric acid with soda powders, and sometimes for cream of tartar. 1. It is easily dissolved by water—it is taken up by fourteen parts of cold, and two of boiling water; the solution has a faint acid reaction, and an acrid metallic taste; it is decomposed and becomes mouldy by long keeping. It is not soluble in alcohol. 2. The powder dropped into *sulphide of ammonium* is turned of an orange or reddish-brown color, and is thereby known from other poisonous metallic salts. 3. *Heated* on mica in air, it is not volatilized, but is charred and evolves the peculiar odor of burnt tartaric acid. When heated in a reduction-tube, it is charred, but does not melt before charring, like the acetate of lead. The metal is partially reduced by the carbon of the tartaric acid, and a peculiar odor is evolved. The decomposed mass has a grayish-blue metallic lustre. No metallic or crystalline sublimate is produced in this experiment by the heat of a spirit-lamp. 4. When boiled in water containing one-sixth part of pure hydrochloric acid, and metallic *copper* is immersed in the boiling liquid, a gray deposit of antimony takes place on this metal. The color of the deposit is violet-red if the quantity is small, but the deposit is iron-gray, like arsenic, or even black and pulverulent, if large. 5. The solution, acidulated with one-tenth part of hydrochloric acid, gives in the cold a black deposit of antimony on a surface of pure *tin*. A slip of tin-foil free from lead should be used in this experiment. A solution of arsenic produces no deposit on tin under these circumstances. Another method of distinguishing antimony from arsenic, or of detecting antimony when mixed with arsenic, is to employ the chloride of tin with fuming hydrochloric acid in equal parts. Both acids of arsenic and all their solid compounds are immediately precipitated on boiling them in this mixture, as brown metallic arsenic. Pure tartar emetic in powder produces no change of color or precipitate, unless it contains some traces of arsenic, when the liquid will acquire a brownish color.

Fig. 37.



Crystals of tartar emetic, magnified 30 diameters.

Tartar Emetic in Solution.—1. On slowly evaporating a small quantity on a slip of glass, it will crystallize in *tetrahedra* and in other derivatives of the octahedron. If obtained from a very diluted solution, the crystallization is prismatic with angular forms. The crystals differ from those of arsenic in decomposing a polarized ray of light. 2. *Diluted nitric acid* added to the solution, throws down a white precipitate (subnitrate of antimony); the other two mineral acids act in the same way; but, as they precipitate numerous other metallic solutions, there are objections to them which do not hold with respect to nitric acid. The white precipitate thus formed possesses the remarkable property of being entirely dissolved by a solution of tartaric acid; it is also soluble in a large excess of nitric acid, so that if much of the test be added at once, no precipitate is produced. 3. *Ferrocyanide of potassium* does not precipitate the solution, whereby tartar emetic is known from most other soluble metallic poisons. 4. *Sulphide of ammonium*, or *sulphuretted hydrogen gas*, produces in the solution a reddish orange-colored precipitate, differing in color from every other metallic sulphide. The dried precipitate is not soluble in ammonia, but is dissolved on boiling by strong hydrochloric acid; and this solution, if too much acid is not present, is again precipitated as a white insoluble oxide of antimony on adding a portion of it to water. The sulphuretted hydrogen is the only test available when the quantity of antimony present in the solution is small. It is the special and characteristic test of the metal. It will reveal the presence of $\frac{1}{500}$th of a grain of tartar emetic; or, under favorable circumstances, the 1000th part of a grain of antimony. There are no objections to the results obtained by this test.

The foregoing tests, it will be observed, merely indicate the presence of oxide of antimony—but this is in reality the poison which we have to seek—the cream of tartar with which it is combined being merely the vehicle; and in a case of poisoning, this is no more the object of medico-legal search than if it were the vehicle for the administration of arsenic or corrosive sublimate. It is, besides, well known that tartar emetic is the only soluble compound of the oxide of antimony which is likely to be met with in medicine or chemistry. Should it be required to prove the presence of cream of tartar, this may be done by filtering the liquid from which the oxide of antimony has been entirely precipitated by sulphuretted hydrogen gas. On evaporating this liquid, the cream of tartar may be obtained as a white crystalline residue.

In Liquids containing Organic Matter.—Tartar emetic is precipitated by tannic acid in all its forms, but not readily by albumen or mucous membrane; therefore it may be found partly dissolved in the liquids of the stomach, provided no antidote has been administered. The precipitates produced with tannic acid and other organic substances are quite soluble in tartaric acid.

Various trial-tests may be used to determine whether antimony is really present in an organic liquid before attempting to separate the metal as sulphide. 1. A few drops poured off in a clear state may be treated with sulphuretted hydrogen gas. If antimony is present in moderate quantity, the fact will be indicated by the production of an orange-red froth and a red precipitate. If the organic liquid is deeply colored and mixed with blood or mucus, a small portion of it may be placed in a tube dialyzer (see *ante*, p. 154), and a clear liquid obtained, in which the precipitated sulphide may be seen of its true orange-red colors. 2. This test, however, may produce no change, and yet the metal may be present. In this case we acidulate a small quantity of the clear liquid with one-tenth of its volume of strong hydrochloric acid, and plunge into it a slip of pure tin-foil. The presence of a soluble compound of antimony is indicated by the rapid deposit of the metal in the form of a black powder upon the tin. 3. The clear liquid acidulated with one-sixth part of hydrochloric acid, and boiled, will, if antimony be present, rapidly deposit the metal of a violet-red color, or as a gray metallic coating on the surface of a bright slip of copper-foil. A negative result from these tests (2 and 3) would show the absence of antimony. (*Guy's Hospital Rep.*, 1860, p. 214.) 4. The galvanic test. A small quantity of the organic liquid may be placed in a platinum capsule and acidulated with a sufficient quantity of hydrochloric acid to hold dissolved any precipitate which may be in the first instance formed. Zinc-foil is then brought into contact with the platinum through the acid liquid, and if antimony is present, the platinum where touched by the zinc, is covered with a black layer of metallic antimony.

If the presence of antimony is indicated by any of these tests, the contents and coat of the stomach, finely cut up, or any solid articles of food, should be boiled in water strongly acidulated with tartaric acid. The mucous and bloody contents of the stomach, with the insoluble deposits found therein, should be similarly treated. The liquid should be strained and filtered from any insoluble matter. A portion of it may be placed in a dialyzing vessel (see *ante*, p. 155), and after a few hours the dialyzed liquid may be found in a clear state for testing. A mouldy and decomposed solution of tartar emetic kept for a year, and mixed with coffee, milk, and albumen, thus speedily yielded a clear and almost colorless liquid in a state fitted for the application of the tests. A current of sulphuretted hydrogen gas may now be passed into the filtered organic liquid until there is no further precipitation. The sulphide is collected, washed, and dried. If it is the sulphide of antimony, it will have an orange-red or brown color, and will, when dried, be dissolved by a small quantity of boiling hydrochloric acid (forming chloride of antimony) with the evolution of sulphuretted hydrogen. The boiling should be continued for several minutes, until all color has disappeared. On adding this solution to a large quantity of water, a dense white precipitate of oxychloride of antimony (powder of Algaroth or Algarotti, *Mercurius Vitæ*) falls down. This is characteristic of antimony. If it be objected that chloride of ***bismuth*** undergoes a similar change when poured into water, sulphide of ammonium will easily enable the operator to distinguish the two metals; the antimonial precipitate is turned of an orange-red color by that solution, while the bismuthic precipitate is turned of a deep black. The antimonial precipitate is easily dissolved by tartaric acid, that of bismuth is not. A soluble compound of bismuth could not have been present in the organic liquid under the circumstances.

That portion of the organic liquid submitted to dialysis, if clear and free from dissolved organic matters, may be concentrated by evaporation. A few drops of the concentrated liquid may yield crystals recognizable by their form or their action on polarized light. The remainder of the liquid may be submitted to the tests elsewhere described for a solution of tartar emetic (*ante*, p. 450), the greater part being reserved for precipitation by sulphuretted hydrogen and the determination of the properties of the precipitate.

It has been a question on a trial of considerable importance in the United States (the case of Mrs. Wharton, Baltimore, 1871–2, see *ante*, p. 111), whether the chemical evidence of the presence of antimony would be complete and satisfactory in those cases in which the analysis was limited to the production of the sulphide, its conversion into chloride, and the action of water upon it. If the preliminary experiments described as trial-tests (2, 3, 4, p. 451) have been performed on portions of the organic liquids, the evidence should be regarded as conclusive and satisfactory. The process would be free from all objections. The antimony would be reproduced as metal by one or all of the methods mentioned.

Supposing these preliminary trials to have been omitted, the chloride of antimony obtained from the conversion of the sulphide might be still made subservient to the production of the metal in various forms. A small quantity of the chloride thus obtained, instead of being added to water (or in addition to this experiment), should be employed for obtaining the metal antimony by copper, tin, or platinum with zinc. Very little of the material will suffice for this corroborative result, and it would show positively that the metal antimony was really present. To rely upon the production of an orange-red or brown precipitate and the action of acids and water on a trial for murder by poisoning with antimony, would be as unsatisfactory as a reliance upon a yellow precipitate and its solubility in ammonia as conclusive evidence of the presence of arsenic, on a trial for murder by poisoning with that substance. No medical jurist would in the present day rest his evidence upon such an incomplete foundation. By the methods described he may readily obtain the metal and convert this into oxide and sulphide, or he may obtain the sulphide and convert this into the chloride and metal. (See *Guy's Hospital Rep.*, 1860, p. 263.) Assuming that the presence of antimony in the contents of the stomach has been satisfactorily demonstrated, it by no means follows that the antimonial compound has been taken or administered as a poison; since it is frequently prescribed as a medicine, and often taken as such by persons of their own accord. We could only suspect that it existed as a poison, or had caused death, when the quantity present was large, and there were corresponding appearances of irritation and inflammation in the alimentary canal. In two cases of criminal administration in small doses, the quantity found in each body did not exceed three grains. The discovery of it in a medicinal mixture would not of itself be evidence of an intent to poison.

Quantitative Analysis.—The quantity of tartar emetic present in a liquid may be determined by the weight of the washed and dried sulphide obtained from a measured quantity of liquid by precipitation with sulphuretted hydrogen. One hundred parts of the dried sulphide by weight are equivalent to 202.78 parts of crystallized tartar emetic.

The facts connected with the absorption, deposition, and elimination of antimony have been already described (*ante*, p. 44). I have found, in accordance with the statements of Orfila, that in poisoning with tartar emetic, antimony is retained by the organs partly in a form soluble in water. Thus a watery extract of the liver in a case of poisoning with antimony, was found to yield traces of the metal, but the proportion deposited is too small to admit of extraction by water, and precipitation by sulphuretted hydrogen. Reinsch's process may be adopted, as the antimony is thereby at once obtained in the metallic state. A few ounces of the liver or other organ, cut into small pieces, should be placed in a mixture of one part of pure hydrochloric acid (first proved to be free from antimony) and seven parts of water. The mixture is boiled in a flask provided with a small funnel (see *ante*, p. 325), and while boiling, successive portions of thin copper-foil, freshly brightened, or of fine copper-gauze, may be introduced. Sooner or later, according to the quantity present, antimony is deposited on the copper, producing a gray deposit, with a reddish-violet or purple tint if the quantity be small, and iron gray or black if comparatively large. If no deposit is observed at first, the liquid must be concentrated on the copper before the inference is drawn that antimony is absent. If the copper remain without any metallic tarnish or deposit upon its surface, there is no antimony present. If it has acquired a metallic deposit, then, after well washing and drying it, further steps must be resorted to in order to determine that it is really antimony with which the copper is coated. Reinsch considered that antimony was sufficiently indicated: 1, by the color of the deposit being violet; and 2, by the copper-foil (when heated) yielding no distinct crystalline sublimate like arsenic; but it was long felt by chemists that these characters, affirmative and negative, were not sufficient for medical evidence. Dr. Odling has suggested the following method of corroboration. (*Guy's Hospital Reports*, October, 1856.) The copper with the supposed antimonial deposit is boiled in a small quantity of water, rendered feebly alkaline by pure potash, and colored of a light crimson or pink tint by the addition of a few drops of a weak solution of permanganate of potash. In a short time the copper loses the whole of the metallic deposit; the liquid becomes colorless, and a brownish substance (hydrated peroxide of manganese) falls down, which should be separated by filtration. A few drops of hydrochloric acid are added to the filtered liquid, and a current of sulphuretted hydrogen passed through it. If the deposit were antimonial, antimoniate of potash would be first formed, and the antimony would be thrown down in the last stage as hydrated orange-red sulphide.

The late Mr. Watson, of Bolton, showed that the permanganate was not necessary; he found that the antimonial deposit on the copper was equally oxidized on boiling it with a weak solution of potash only, the metal being partly exposed to air by drawing it out of the alkaline liquid, and then again returning it. In about five or ten minutes the copper will have lost the deposit, and the liquid may then be filtered, acidulated with hydrochloric acid, and treated with sulphuretted hydrogen. The orange-red sulphide of antimony, of its characteristic color, is thrown down either immediately, or on allowing the liquid to stand for a few hours. The dried sulphide thus obtained from the deposit may now be dissolved in strong hydrochloric acid, and having prepared a small Marsh's tube with pure zinc and water (see Fig. 25, *ante*, p. 323), the chloride may be poured by a funnel-tube into the apparatus, and the escaping gas dried by chloride of calcium, as in the operation on arsenic. Antimonuretted hydrogen, which is liberated under these circumstances, possesses the following properties: 1. If the gas is allowed to pass over a solution of nitrate of silver on paper, it blackens it, and sets free metallic silver; 2. If kindled it burns with a pale, lemon-colored flame, evolving a white smoke (oxide); 3. A piece of white porcelain acquires, when placed so as to intercept the flame about its centre, a deep coal-black deposit, with gray rings. The coal-black color distinguishes this deposit from that of arsenic, which is hair-brown. In addition, the antimonial deposit is not dissolved by a solution of chloride of lime, while the arsenical deposit is dissolved; 4. If the gas is allowed to escape through a reduction-tube drawn out as in Fig. 25, p. 323, and the tube is heated to redness, a deposit of metallic antimony will take place at or near the point heated. This is of a lighter color than the deposit of arsenic; it yields no octahedral crystals when heated, and is not dissolved by a solution of chloride of lime. There is another important distinction. When the gas is made to pass through a small quantity of fuming nitric acid containing nitrous acid, it is decomposed, the antimony is peroxidized, and may be obtained as a white, insoluble residue on evaporation. A solution of nitrate of silver produces no change of color in this deposit; but if one or two drops of ammonia are added, there is a black precipitate of antimonide of silver. Arseniuretted hydrogen similarly treated produces arsenic acid, which gives a red precipitate with a solution of nitrate of silver with or without ammonia. The absence of arsenic from the dissolved chloride may, however, be proved before placing it in the Marsh's tube. This may be effected by boiling a few drops of the chloride with a solution of the chloride of tin and hydrochloric acid. If chloride of arsenic is present, this will be indicated by a brown deposit. If there is no change of color or deposit, then no arsenic is present. In this case the gas evolved would be the antimonuretted hydrogen.

By another process we may obtain in the first stage the metal, and convert this into the chloride and sulphide.

The acid liquid obtained by boiling the liver or other organ in hydrochloric acid may be concentrated by evaporation, and placed in a platinum capsule. By the aid of zinc the antimony will be deposited on touching the platinum through the liquid. (See *ante*, p. 451.) The metal goes down as a black powder, closely adhering to the platinum. It should be well washed with distilled water, then treated with strong nitric acid, which converts it into peroxide of antimony. This remains as a white deposit in the capsule on evaporating the nitric acid. It may now be readily dissolved by warming it with a strong solution of tartaric acid, or with a small quantity of hydrochloric acid. The tartrate or chloride of antimony thus formed may be then converted into the orange-red sulphide by a current of sulphuretted hydrogen gas.

If the quantity of antimony is very small, so that a deposit is not immediately produced, the galvanic process recommended for corrosive sublimate, substituting platinum for gold, may be adopted. (See *ante*, p. 374.) A slip of platinum-foil wound in a spiral round a portion of zinc may be suspended in the liquid for some hours. The liquid must be so diluted as not to act too violently on the zinc. After a time the antimony will be found deposited on the platinum in the form of a black powder. It will also be partly deposited on the zinc. The platinum, with its deposit separated from any remains of the zinc, should be well washed with water, and treated with nitric and tartaric acids, according to the method above mentioned. The amount of *absorbed* antimony found deposited in the organs is always small. In general, from one grain to three or four grains would probably be the whole amount that could be separated by chemical processes from those organs and parts of the body which are usually submitted to analysis.

Although the detection of antimony in the tissues does not necessarily indicate that it has been criminally administered, or has caused death, yet its presence should be reasonably accounted for, as antimony is not a normal constituent of the body, and it may have been secretly and unlawfully administered. In several cases of suspected death from poison, deposits on copper, evidently of an antimonial nature, have been obtained from the liver or tissues. On inquiry it has been found that antimonial medicines had been taken shortly before death. Conversely, when no antimony is found, or the metal is found in the tissues in minute quantity, it is still consistent with medical experience and observation that the person may have died from antimony. The case of Mrs. Peters, of Yeovil (July, 1860), furnishes a remarkable illustration of this fact. This lady had symptoms during her illness which were referred by her medical attendants to the effects of small doses of antimony. Antimony was found in the urine both by them as well as by Mr. Herapath; but after death (*i. e.*, in about nine days) no antimony was found in the tissues or any part of the body. Upon this fact and the evidence of coexisting disease, it was alleged that she had died from disease and not from poison. The jury returned a verdict to the effect that her death had been accelerated by irritant poison. (*Med. Times and Gazette*, Aug. 25, 1860, p. 190; Sept. 15, p. 271; and Sept. 29, p. 317.) Assuming the results of the analysis of the urine during life to have been correct, there can be no question that antimony was administered to her; and the statement of the acceleration of death is rendered probable. The case is important in this respect: it shows that antimony may be found in an evacuation, and that death may be accelerated by it; but although the person may die within nine days, none may be detected in the body.

The purity of the acid employed in these processes should be always determined. Hydrochloric acid frequently contains arsenic, as an impurity, but antimony is rarely found in it. In the year 1856, a sample of this acid was sent to me, which contained so much antimony, that on mixing it with water, it gave an abundant precipitate of oxychloride. The acid had been employed with carbonate of soda in making unfermented bread, and it had produced in a provincial town a large amount of sickness, the cause of which could not at the time be explained.

Detection after long Periods.—Antimony, even in a soluble form, does not readily disappear from a dead body after interment. If a person dies with absorbed or free antimony in his body, some portion of the metal may be extracted, probably so long as any portions of the viscera remain. In the case of Ann Palmer, the body was exhumed after an interment of *fourteen months*, and antimony was found in the free state in all parts of the alimentary canal, and in the absorbed state, more or less in all the organs. One ovary alone yielded the fiftieth part of a grain. The antimony had partially undergone a chemical change, as a result of putrefaction. In the stomach, a portion of it had been converted into orange-red sulphide, which dyed the coats in a streak or stain from the inside to the outside. In the rectum it was also partially changed into sulphide. In this case, antimony manifested an antiseptic property like arsenic, for all the parts in which the metal was found were well preserved. In the case of Ann Bacon, whose body was exhumed in 1857, after *twenty-one months'* interment, antimony was found in the intestines. The presence of the metal was here traced to some small doses of antimonial medicine which had been given to the deceased during her last illness, and shortly before her death. Arsenic is occasionally present as an impurity in antimonial compounds. It may be readily detected by Bettendorff's test (*ante*, p. 306).

CHLORIDE OF ANTIMONY—SESQUICHLORIDE OR BUTTER OF ANTIMONY.

This is a highly corrosive liquid, varying from a light yellow to a dark red color—in the latter state containing generally a large quantity of chloride of iron. It is a powerful poison, but it is not often taken as such. Orfila mentions only one, and that a doubtful instance, which occurred nearly two hundred years ago. I have collected reports of several cases, in three of which recovery took place.

Symptoms.—A boy, æt. 12, swallowed by mistake for ginger-beer four or five drachms of a solution of butter of antimony. In half an hour he was seized with vomiting, which continued at intervals for two hours. There was faintness, with general weakness and great prostration of strength. Remedial means were adopted, and the next day the chief symptoms were heat and uneasiness in the mouth and throat, with pain in swallowing. There were numerous abrasions on the mucous membrane of the mouth and fauces; and there was slight fever, from which the boy quite recovered in about eight days.

In a second case, about a tablespoonful of chloride of antimony was given, by mistake for antimonial wine, to a boy, æt. 10. Immediately on drinking it, the boy seemed choked; his features were set, and he was unable to speak for some minutes. He then vomited freely; gruel was given to him, which was rejected; he complained of great pain in his throat. Medical assistance was sent for, and about two hours after swallowing the poison, he labored under the following symptoms: The features were pale and collapsed, the eyes sunk, the pupils dilated and inactive, the skin cold, the mouth filled with a thick, tenacious, transparent mucus; nausea, vomiting, pulse 80 and small, and breathing heavy. He was in a kind of stupor, from which he could, however, be roused to answer questions rationally. He felt a severe burning pain in the throat, extending to the stomach, increased by the act of swallowing. Under active medical treatment, these alarming symptoms were removed; on the following day it was observed that there were patches of a bright scarlet color in the throat, with some difficulty of swallowing. In the course of a few days the boy recovered. The following case occurred to Mr. Bancks, of Stourbridge: A boy, æt. 7, swallowed two drachms of chloride of antimony, sent in mistake by a druggist, who immediately discovered his error, and applied for medical assistance. There was excoriation of the mouth and throat; the skin was cold and clammy; pulse small and accelerated; burning pain in the stomach, with swelling of the abdomen, and incessant vomiting. Magnesia diffused in water, a decoction of bark and strong tea, were given at intervals until 8 P.M., when there appeared much less pain in the stomach. The boy gradually recovered, and in four days was out of danger. For the next few days he continued to improve, and was soon in perfect health again. It is worthy of remark that the child had taken no food on the morning he swallowed the poison—a circumstance much against the chance of recovery. (*Prov. Med. Jour.*, Dec. 23, 1846.) Another case of recovery from a dose of an ounce is reported in the *Lancet*, Feb. 26, 1848, p. 230.

An army surgeon swallowed, for the purpose of suicide, from two to three ounces by measure of chloride of antimony. About an hour afterwards he was seen by Mr. Mann. There was entire prostration of strength, with coldness of the skin, and incessant attempts to vomit. Severe griping pains were felt in the abdomen; and there was a frequent desire to evacuate the bowels, but nothing was passed. In the course of a few hours reaction took place, the pain subsided, and the pulse rose to 120. There was now a strong disposition to sleep, so that he appeared as if laboring under the effects of a narcotic poison. In this state he continued until he died—ten hours and a half after he had swallowed the poison.

Appearances after Death.—The interior of the alimentary canal, from the mouth downwards to the middle of the small intestines, presented, in the abovementioned case, a black appearance, as if the parts had been charred. In general, there was no mucous membrane remaining, either in the stomach or elsewhere; only a flocculent substance, which could be easily scraped off with the back of a scalpel, leaving the submucous tissues and the peritoneal coat. All these parts were so soft that they were easily torn with the fingers.

Mr. Evans, of Northampton, has given me in detail a case which occurred to him in May, 1868. A man swallowed three or four ounces of bronzing liquid, which proved to be a solution of chloride of antimony. He vomited violently, but continued his work for an hour; the vomited matters were of a yellowish-green color. There was pain in the stomach, but no purging. He was not seen by a medical man. He had passed a sleepless night, and complained much of oppression in the region of the heart. He died in about eighteen hours. On inspection, the mucous membrane of the stomach was found much corroded. Near the intestinal end there were numerous putty-like masses. In parts it was of a vividly red color, and in other parts blackened. There was no perforation. The duodenum presented similar appearances. There was no mark of corrosion on the lips, or on the lower part of the gullet. The upper part of this tube, the fauces, and mouth, could not be examined. Antimony was found in the putty-like masses of membrane, as well as in the contents of the stomach and in the liquid swallowed.

Treatment.—The free exhibition of magnesia in milk, as well as of infusions containing tannic acid, or a solution of the acid itself.

CHEMICAL ANALYSIS.

If any portion of the chloride is left in the vessel, it may be tested by adding a few drops to water, when the whitish-yellow oxychloride of antimony will be precipitated; the supernatant liquid will contain hydrochloric acid, which may be detected by nitrate of silver. It has been already observed, that the only objection to this mode of testing is, that the salts of *bismuth* are also decomposed by water; but the precipitate in this case is insoluble in tartaric acid, and is blackened by sulphide of ammonium; while in the case of antimony, the white precipitate is soluble in this acid, and is changed to an orange-red color by the sulphide. The precipitate oxide of antimony is soluble in potash; and on adding nitrate of silver to the potash-solution, a dense black precipitate is formed insoluble in ammonia. Nitrate of silver added to the chloride gives a mixed precipitate of chloride of silver and oxide of antimony, the former being soluble in ammonia. If the chloride contains much iron, the true color of the precipitate will be obscured. Ferrocyanide of potassium has no effect on a solution of tartar emetic, but it precipitates the chloride of antimony of a yellow-white; or if much iron is present, Prussian blue will be abundantly thrown down.

Organic Liquids or Solids.—The chloride, as a corrosive, combines with the animal tissues. The antimony may be separated in such cases by boiling them in tartaric acid. The filtered liquid may then be further tested by a current of sulphuretted hydrogen, as well as by the processes of Marsh or Reinsch (p. 454).

In the Tissues.—Any antimonial compound may be dissolved out of the coats of the stomach or other structures, by boiling them in hydrochloric acid slightly diluted. The hydrochloric solution, when cold, may be further diluted with nine parts by measure of water, and a slip of pure tin-foil suspended in the liquid. A black deposit on the tin, after sufficient contact, indicates the presence of antimony. The metal may be also separated by platinum and zinc, according to the method described at p. 451, or it may be precipitated by sulphuretted hydrogen.

CHAPTER XLIX.

POISONING WITH THE SALTS OF ZINC, SULPHATE, AND CHLORIDE—SYMPTOMS—APPEARANCES—BURNETT'S LIQUID—CHRONIC POISONING—TREATMENT—ANALYSIS—ORGANIC LIQUIDS AND THE TISSUES.

COMPOUNDS OF ZINC—SULPHATE OF ZINC—WHITE VITRIOL.

Symptoms and Appearances.—The symptoms produced by an overdose of sulphate of zinc, are pain in the abdomen and violent vomiting, coming on almost immediately, and followed by copious purging, and great prostration of strength. After death, the stomach has been found inflamed. The sulphate appears to act as a pure irritant; it has no corrosive properties. This salt may cause death indirectly as the result of exhaustion from violent vomiting and purging, when a large dose has been given to a person already debilitated by disease. (*Med. Times and Gaz.*, July 16th, 1853, p. 78.) Dr. Gibb has reported a case of poisoning by this substance, in which a lady recovered after taking sixty-seven grains. (*Lancet*, May 17th, 1856.)

Tommasini relates that a lady swallowed, by mistake, one ounce of sulphate of zinc. Among the first symptoms were violent pains in the stomach, vomiting, and convulsions, in fact, severe irritation. The pain continued for some time. There was paleness of the countenance, coldness of the limbs, irregular pulse, cold sweats, and fainting. This state of collapse was alarming. Stimulants were given. At night, besides pain in the abdomen and unnatural heat, there was fever, with other symptoms of reaction. Inflammation of the stomach supervened. It was some time before recovery took place. (*Della nuova dottrina med. Italiana*, 1817, p. 57.)

It is rare that this substance proves fatal. It has no local action, and, as it acts as an emetic, it is speedily ejected from the stomach. In May, 1872, a case occurred to Dr. Mackintosh, of Downham, in which a man, æt. 20, recovered in a few days after taking an ounce of sulphate of zinc, by mistake for Epsom salts. There was early vomiting and purging, but of a most violent kind, followed by great prostration of strength. The greater part of this large dose had been thus carried out of the body. In a case which occurred to Dr. Ogle, the sulphate destroyed life by its slow or chronic effects. (*Lancet*, Aug. 27th, 1859, p. 210.) Neither the sulphate nor the oxide of zinc can be regarded as powerful irritants, although they are usually described as poisons. MM. Tardieu and Roussin have published a case of criminal poisoning by sulphate of zinc administered in soup. A woman, æt. 60, died in three days under the usual symptoms of irritant poisoning (gastro-enteritis). Zinc was detected in the coats of the stomach and intestines, as well as in the spleen and liver. (*Ann. d'Hyg.*, 1871, pp. 2, 341.) In one case a lady recovered after taking sixty-seven grains. (*Lancet*, May 17th, 1856.) In cases of epilepsy, the late Dr. Babington gave sulphate of zinc in doses of two scruples, three times a day, having first commenced with small doses. He gave to a girl, æt. 17, thirty-six grains of the sulphate three times a day for several weeks, without any sickness or other outward effects being produced. When the dose was raised to forty-two grains, which the girl continued to take for a week, she lost her appetite and felt much sickness. (*G. H. Reports*, No. 12, p. 17.) This may have been owing to a tolerance of the medicine. With respect to the *oxide of zinc*, Dr. Marcet states that he has prescribed it in large doses without injury to health. One patient, an epileptic, took as much as one pound in seven months, the largest quantity taken in one day being seventy grains. Although he did not suffer from the remedy, the disease was not cured. (*Lancet*, March 1st, 1862, p. 224.)

Treatment.—Tepid water, with milk or albumen, should be freely given to promote vomiting. Any infusions containing tannic acid may be employed, such as tea, oak-bark, or Peruvian bark. The stomach-pump may be used. If the poison should have entered into the intestinal canal, a fact indicated by severe pain in the lower part of the abdomen, emollient enemata may be administered.

Chemical Analysis.—The pure sulphate is seen in colorless prismatic crystals, resembling in appearance sulphate of magnesia and oxalic acid; from oxalic acid it is distinguished by remaining fixed when heated on platinum-foil; from the sulphate of magnesia, by tests applied to its solution. It is readily dissolved by water, this fluid taking up about one-third of its weight at common temperatures Tests for the *solution:* The solution in water has a slightly acid reaction. The following tests may be used for the detection of oxide of zinc: 1. *Ammonia* gives a white precipitate, soluble in an excess of the alkali. 2. *Sesquicarbonate of ammonia*, a white precipitate, also soluble in a large excess of the test. 3. *Ferrocyanide of potassium*, a white precipitate. 4. *Sulphuretted hydrogen* and sulphide of ammonium, a milky-white precipitate, provided the solution be *pure* and neutral, or nearly so. If the solution is very acid, sulphuretted hydrogen produces no effect whatever. 5. Zinc may be separated in the metallic state by placing in the solution a slip of magnesium. Nitrate of baryta will serve to indicate the presence of sulphuric acid.

In Organic Liquids.—If the sulphate of zinc is dissolved and the solution is not too acid, we may pass into it a current of sulphuretted hydrogen gas; the presence of zinc is immediately indicated by a milky-white froth and precipitate; the sulphide may be collected and decomposed by boiling it with hydrochloric acid. The solution may be then tested for zinc. If the organic liquid is of a viscid nature, and highly colored, it should be acidified and placed in a dialyzing vessel (*ante*, p. 155.) We thus speedily obtain in a clear and almost colorless state, a solution to which all the tests can be applied. The sulphate is frequently employed as an emetic, and may be innocently present in an organic liquid, or in the contents of the stomach. It has been occasionally used as a fraudulent addition to bread. (Horn's *Vierteljahrsschrift*, 1870, pp. 1, 323.)

In the Tissues.—If we have to search for zinc in the mucous membrane of the stomach, or in the substance of the liver, these may be cut up and boiled in strong nitric acid. The viscera may be also incinerated with flux, and the zinc procured in a metallic state, or dissolved out of the residue by hydrochloric acid.

CHLORIDE OF ZINC.

This is a solid white uncrystalline compound, very soluble in water, and possessing corrosive as well as irritant properties.

A solution of it, in an impure state, is sold to the public under the name of Sir W. Burnett's disinfecting fluid. It has been taken by accident or mistake in many cases, and has been the cause of numerous deaths. It is either colorless, like water, and has then been fatally mistaken for "fluid magnesia," or it is of a yellowish color from the presence of oxide of iron, and has then been mistaken for pale ale. In August, 1856, it gave rise to a fatal accident in one of the American steamers. It was served to the Rev. T. Marsh, a passenger, by mistake for mineral water. It is stated that he did not swallow more than a mouthful, as he immediately perceived a burning sensation in his throat. He died from the effects of the poison on the fourth day! Most of these accidents have occurred from gross carelessness in keeping this noxious fluid in ordinary wine or medicine bottles, and in proximity to innocuous liquids which resemble it.

Symptoms.—The symptoms come on immediately. In a case reported by Dr. Stratton, about two ounces of a solution containing only twelve grains of the chloride were swallowed. The patient immediately felt pain and nausea; vomiting followed, and she recovered, but suffered from some indisposition for three weeks. In a second case, a wine-glassful, equivalent to at least two hundred grains of solid chloride, was swallowed. The man instantly experienced burning pain in the throat, burning and griping pain in the stomach, great nausea, and coldness. Vomiting came on in two minutes; the legs were drawn up to the body; there was cold perspiration, with other signs of collapse.

The man perfectly recovered in sixteen days. (*Ed. Med. and Surg. Journal*, Oct. 1848, p. 335; and *British American Journal*, Dec. 1848, p. 201.) Other cases show that the concentrated liquid has a strong corrosive action locally, destroying the membrane of the mouth, throat, gullet, and stomach. There has been frothing of the mouth, with general lividity, coldness of the skin, and other signs of collapse. In a case in which only a mouthful, *i. e.*, from four to six drachms of the fluid, had been swallowed, the patient experienced giddiness and loss of sight, with immediate burning heat in the stomach; vomiting and purging came on, and the former symptoms continued for a week. There was so much irritability of the stomach for a period of three weeks, that the patient was greatly reduced. Among the early symptoms was loss of voice, which did not return for five weeks. (*Med. Times*, Oct. 11, 1851, p. 382; and Nov. 8, 1851, p. 497.) Dr. R. Hassall met with a case in which the nervous symptoms were strongly marked, and were of a peculiar kind. Three ounces of "Burnett's Fluid" were swallowed. There was immediately a sense of constriction in the throat, with a hot burning sensation in the stomach. It is worthy of remark, that there was no pain in the mouth, and there was no appearance of corrosion in this cavity or on the lips. This absence of local chemical action was noticed in another case, reported by Dr. Tuckwell, of Oxford. (*Brit. Med. Jour.*, Sept. 5, 1874, p. 297.) There was incessant vomiting, the vomited matters consisting of thick mucus streaked with blood, and some portion of mucous membrane was discharged. There was no purging until the third day, when the discharges from the bowels had a coffee-grounds appearance. After the lapse of a fortnight, a train of nervous symptoms set in, indicated by a complete perversion of taste and smell. The patient appears to have recovered in about three months. (*Lancet*, Aug. 20, 1853, p. 159.)

Two cases, reported by Mr. E. Aikin, show that the severity of the symptoms is not always to be measured by the quantity taken. A gentleman swallowed by mistake for Friedrichshall water, four drachms of Burnett's solution, corresponding to about fifty grains of the solid chloride of zinc. He immediately felt a sense of strangulation and burning in the throat and stomach. Vomiting was induced by olive oil and milk and water. In half an hour he complained of severe pain in the throat and stomach. The vomiting was incessant, but there was no sign of collapse. Albumen was given, but this was quickly ejected in a coagulated state. The vomiting slowly subsided; there was no purging, but there was great tenderness in the region of the stomach. The pulse did not rise above 120. On the following day the vomiting had nearly ceased, but there was great tenderness in the abdomen, and the throat was very red. In a week he recovered.

A female servant was attacked on the same day and hour with symptoms of poisoning. She had tasted the contents of the glass, but did not swallow more than a teaspoonful. The symptoms were unusually severe, as vomiting could not be induced until an hour had elapsed. She was much purged, and at one time appeared to be in a state of collapse. Pain was referred to one small spot in the epigastric region, and she suffered uneasiness there every time food was swallowed. She recovered; but the poison had no doubt, in this case, exerted a corrosive action upon the gastric mucous membrane. (*Guy's Hosp. Gazette*, Dec. 6, 1873, p. 74.)

Mr. Allanson, of Sheffield, communicated to me the following case: A woman, æt. 28, swallowed an ounce of a strong solution of the chloride, which had been sold to her as disinfecting fluid. In two hours she was lying on her back in a state of great excitement. The face was flushed, the eyes were turned, a frothy saliva was issuing from the mouth, the hands and feet were cold, and the pulse was scarcely perceptible. She was perfectly conscious, and complained of a burning sensation in the mouth, throat, and stomach. The tongue was found swollen, and the mucous membrane red, but there was no excoriation. It was at first supposed that she had swallowed oil of vitriol. In spite of treatment, she died in *four hours* after she had taken the poison. While she survived there was frequent vomiting, but the most prominent symptom throughout was severe pain in the stomach and throat.

A lady swallowed three parts of a wineglassful of Burnett's liquid by mistake. In twenty minutes there was violent vomiting of a mucous and bilious liquid. The countenance was dusky and anxious; the pulse quick and fluttering (130), and there was a sense of burning pain in the œsophagus. The pupils were small, the skin moist, and there was great prostration of strength. She died in seventeen hours after taking the poison. A girl, æt. 17, swallowed half a wineglassful of the fluid, and died from the effects in less than *two hours*. The symptoms here were copious vomiting of frothy mucus with shreds of membrane, and cramps in the legs, which were drawn up to the abdomen. Other cases, in which the symptoms and appearances were somewhat similar, will be found reported in the *Lancet*, 1864, vol. 1, p. 35. In May, 1864, a woman, æt. 63, swallowed one ounce and a half of Burnett's solution. She almost instantly experienced great pain in the stomach, and vomited freely. Shortly afterwards, she was much purged. Albumen and mucilage were given. An hour and a half after taking the poison, she was much collapsed, with cold limbs, clammy sweats, blistering of the lips and tongue, pulse very small and quick. She complained of burning pain in the throat and stomach, of giddiness, and loss of sight. Vomiting and purging continued. She had lost her voice. At this time the vomited matters contained mucus but no blood. The motions were thin and dark brown. During the night she had occasional slight fits—losing consciousness, and having twitchings of the facial muscles. She did not rally. Vomiting and purging continued up to the time of death, 15½ hours after taking the poison. (*Lancet*, Sept. 3, 1864, p. 267.)

The following are cases of *chronic* poisoning. One of them, which occurred to Dr. Markham, proved fatal in about *ten weeks* after the poison had been swallowed. The patient, a woman, æt. 46, took half a wineglassful of Burnett's liquid, equal to about 100 grains of chloride of zinc. Immediately after taking it, she suffered from vomiting and pain in the stomach. She drank freely of water; the vomiting ceased in a few days, and she appeared to have recovered. In about three weeks the vomiting returned; it was incessant, and with this, there was pain in the stomach. She sank exhausted, evidently from the secondary effects of the poison. (*Medical Times and Gazette*, June 11, 1859, p. 595.) In 1863 several deaths were reported to have taken place in consequence of Burnett's fluid having been mistaken for medicine. In one of these a lady swallowed a wineglassful in place of fluid magnesia. She suffered severely, and died after six weeks from the secondary consequences of the poison.

Appearances after Death.—Out of ten cases of which I have collected the particulars, there have been six deaths. In one, an infant, æt. fifteen months, the lining membrane of the mouth and throat was white and opaque. The stomach was hard and leathery, containing a liquid like curds and whey. Its inner surface was corrugated, opaque, and tinged of a dark leaden hue. The lungs and kidneys were congested. The fluid of the stomach contained chloride of zinc. (*Med. Times*, July 13, 1850, p. 47.) A seaman, æt. 21, swallowed about half a pint of Burnett's solution. The case proved fatal in spite of treatment, and the appearances (twenty-five hours after death) were as follows: The neck was swollen and the hands were clenched. There was great lividity of the body and arms. The stomach externally was reddened; the mucous membrane was of a deep purple color, and partially corroded and destroyed. The pyloric or intestinal opening was constricted, and the mucous membrane at this part looked as if caustic had been applied to it. The upper part of the gullet was constricted and of a purplish color; the mucous membrane of the remainder was covered with a fine false membrane, and there was a loose coagulum of recently exuded lymph in the centre. The upper part of the small intestines (duodenum) was of a purplish color, and intensely red for about six inches. There were some red patches in the jejunum, and small emphysematous patches near the lower part of the small intestines. The lungs were congested, and the right lung contained a bloody serum. The heart was normal; the ventricles were filled with dark coagula of blood. In the head, the membranes (dura mater) contained more blood than usual, and there was a general congestion of vessels on the surface of the brain, as well as at the base, The gray matter was darker than usual, and the right choroid plexus was enlarged. (Dr. J. Rose, *Lancet*, Sept. 12, 1857, p. 271.) In the case of the woman, æt. 63 (*supra*), who died in 15½ hours, from the effects of this poison, the body was inspected forty-eight hours after death, and the following appearances were observed. Slight cadaveric lividity of the abdomen and back. The mucous membrane of the lips and tongue was abraded; that of the gullet was entirely destroyed, except in some parts about the lower third of its extent, where it was softened and hung in shreds. The mucous membrane of the epiglottis was abraded, and there was swelling and congestion of the throat and larynx. The peritoneum was injected, but there was no lymph or serum effused. The stomach was of a slate color externally; the veins were much enlarged, and the coats thickened and of a leathery consistency. Internally, the mucous membrane was of an ash color, corrugated and much destroyed. There was no appearance of ulceration or perforation. The duodenum and intestines were congested. Heart normal, the left side gorged with blood. The lungs were congested. (*Lancet*, Sept. 3, 1864, p. 267.) In Mr. Allanson's case (*supra*) the body was examined fifty-three hours after death. The stomach was much distended, of a pale leaden hue, and the veins were dark and prominent. The under surface of the liver, where it was in contact with the stomach, had the same appearance. The intestines and the other viscera were healthy. The stomach contained a quantity of fluid. The coats were of the consistency of thin tripe, and were much thickened at the intestinal end. None of the fluid taken had passed through the pylorus. Well-marked papillæ of a dirty white color covered the whole mucous membrane. The gullet was much inflamed. The epithelial membrane was white and easily detached, appearing like a false membrane. The lungs and heart were perfectly healthy. Chloride of zinc was detected in the stomach. An acute case, which proved fatal in eight hours, is reported in Beale's *Archives of Medicine*, 1858, No. 3, p. 194.

These facts show that the concentrated solution of chloride of zinc is both a corrosive and an irritant poison, exerting also a powerful action on the nervous system. In a case which proved fatal in Guy's Hospital, in 1856, the coats of the stomach were excessively thickened, and had a leathery consistency. The stomach was remarkably contracted—the mucous membrane puckered and of a slate color. There was a perforation in the coats in two places, one at the cardia, the other at the pylorus. The woman had swallowed a wineglassful of Burnett's fluid by mistake for gin. She kept it on her stomach for ten minutes, and then vomited. She had no pain in the stomach, but a burning sensation in the throat and chest. She survived the effects of the poison sixteen days. (*Guy's Hosp. Rep.*, 1859; p. 128.)

In another case, the stomach is described as being shrivelled up and ulcerated. (*Pharm. Jour.*, Jan. 1867, p. 420.) In Dr. Markham's case (p. 463), the stomach was so constricted at the intestinal end by cicatrix, that it would only admit a crowquill. The pyloric opening was involved in this cicatrix, which was about one-quarter of an inch wide. There was no other sign of disease in the body. This case proves that death may occur from the poison even after apparent recovery. The chloride of zinc may destroy life either by producing stricture of the gullet or pylorus, or by its chemical action on the lining membrane of the stomach leading to a loss of power of digestion, emaciation, and exhaustion. Under these circumstances the person really dies of starvation from the chemical action of the chloride on the stomach and bowels. A most instructive case of this kind occurred to Dr. Tuckwell. A girl, æt. 21, swallowed four ounces of Burnett's liquid. She suffered from the usual symptoms, and survived the effects of the poison 117 days, and for the last 57 days was supported only by injections, nothing but water being taken by the mouth. For a minute account of symptoms and appearances, I must refer the reader to the original report (*Brit. Med. Jour.*, Sept. 5, 1874). The condition of the stomach and intestines sufficiently accounted for the state of inanition under which the patient sank.

Treatment.—The free use of albumen, milk, and emetics.

Analysis.—The chloride may be detected by nitrate of silver; the zinc by the tests above described. (See p. 460.) If a portion of the solution is placed in a platinum capsule, and the platinum is touched with magnesium, the zinc is immediately separated in the metallic state. All the samples which I have examined, with one exception, contained iron.

The chemical properties of Burnett's fluid became a subject of inquiry in *Richards* v. *Cocking* (Guildhall Summer Sittings, 1858). The plaintiff charged the defendant, a druggist, with having supplied him with Burnett's fluid in place of fluid magnesia. There was medical evidence that the plaintiff had suffered from such symptoms as chloride of zinc would produce, and he obtained a verdict. On this occasion I examined the liquid usually sold as Burnett's fluid. I found that it had a sp. gr. of 1.494; that it had an oily consistency, and was frothy when shaken. It coagulated albumen even when much diluted, and strongly corroded animal matter. One fluid ounce of it by measure contained 372 grains of solid chloride of zinc. It is a most dangerous substance, and is liable to be fatally mistaken for numerous innocent medicines.

In the Tissues.—Chloride of zinc may be detected in the tissues by the process of incineration in a porcelain capsule, and digestion of the ash in water. It is important to bear in mind that this salt is occasionally used for embalming or preserving dead bodies; hence the discovery of it is of itself no proof of poisoning.

Carbonate of Zinc.—This compound does not appear to have any poisonous action; and it would probably require to be given in large quantity to produce any effect. It is the white substance which is formed on the metal when long exposed to air and moisture. Its effects may become a subject of investigation as a matter of medical police, since zinc is now much used for roofing, and also in the manufacture of water pipes, cans and cisterns. (See *Ann. d'Hyg.*, 1837, p. 281; vol. 2, p. 352; also *Edinburgh Monthly Journal*, Aug. 1850, p. 181.) Its effect on water used for drinking purposes has become a sanitary question, especially since the metal has been employed for the making of cisterns.

CHAPTER L.

POISONING WITH THE COMPOUNDS OF IRON—SULPHATE AND MURIATE—SUBNITRATE OF BISMUTH—PEARL-WHITE—ARSENIC IN BISMUTH—CHROMIUM COMPOUNDS—BICHROMATE OF POTASH—CHROMATE OF LEAD—COMPOUNDS OF THALLIUM—POISONOUS COMPOUNDS OF OTHER METALS—OSMIC ACID.

COMPOUNDS OF IRON.

Sulphate of Iron—Copperas.—This compound has been on several occasions administered with malicious intention. One death was caused by it in 1837-8, and another, which occurred in France, was the subject of a criminal trial in 1869. A man was convicted of having killed his wife and his son by administering to them sulphate of iron in coffee. (Bouchardat, *Ann. de Thérap.*, 1872, p. 146.) It is not, however, an active irritant. A girl who swallowed an ounce of it recovered, although she suffered for some hours from violent pain, vomiting and purging. (Christison, *On Poisons*, p. 506.) It is commonly sold under the name of green vitriol or copperas. It is sometimes given as an abortive. A suspicious case is reported, in which a woman, far advanced in pregnancy, but enjoying good health, was suddenly seized at midnight with vomiting and purging, and died in about fourteen hours. The body, which had been buried, was disinterred, and iron was found in large quantity in the viscera. The symptoms are not always of this violent kind. In a case which occurred to M. Chevallier, a man gave a large dose of sulphate of iron to his wife. There was neither colic nor vomiting. The woman lost her appetite, but ultimately recovered. In another case, reported by the same authority, a woman was tried and convicted of poisoning her husband with sulphate of iron; but in consequence of the great diversity of opinion among the scientific witnesses at the trial, respecting the poisonous properties of this mineral salt, and the dose in which it would be likely to operate injuriously, the court and jury recommended that the sentence of death should not be carried into execution. (*Ann. d'Hyg.*, 1851, vol. 1, p. 155; *Med. Gaz.*, 1850, vol. 45, p. 640.) The reader will find some additional remarks in reference to the action of the sulphate of iron on the body, by Orfila, in the same journal, 1851, vol. 2, p. 337. At the Nottingham Autumn Assizes, 1859, a woman of the name of Riley was indicted for administering copperas to two children. She put the substance into gruel. It gave to the gruel a greenish color and a peculiar taste, which led to the discovery. It caused sickness, but no other serious symptoms. As there was no evidence of an intent to murder, and it was then not unlawful to administer poison with any other intent, the prisoner was acquitted. This salt has been much used for criminal purposes in France. (See *Med. Gaz.*, vol. 47, p. 307; also *Ann. d'Hyg.*, 1850, vol. 1, pp. 180, 416; and 1851, vol. 1, p. 155; vol. 2, p. 337.) Sulphate of iron is said to have proved fatal to sheep. It had been mixed with the pulp of beet-root for cattle-food. (*Med. Times and Gaz.*, 1863, vol. 1, p. 511.)

External Application.—A case which seems to show that this substance may really act through the skin, has been reported by Mr. Moore, of York. A healthy boy, æt. 14, after having been employed in picking crystals from the vat in which sulphate of iron was set to crystallize, was attacked with headache and sickness. He vomited several times, felt pains in the calves of his legs, and colicky pains in the abdomen; at the same time his limbs became contracted. The boy had previously complained that the liquor of the crystals, into which he was constantly dipping his hands, had cracked his fingers. In the course of a week or ten days, these symptoms disappeared under treatment. (*Med. Gaz.*, vol. 30, p. 351.) No other cause could be assigned for this singular attack than the frequent contact of the hands with a saturated solution of the green sulphate of iron.

Chemical Analysis.—This substance is generally met with in crystals of a sea-green color. It is readily soluble in water. 1. *Ferrocyanide* *of potassium* added to the solution, gives a greenish-blue precipitate, becoming of a deep blue by exposure to the air. 2. *Sulphide of ammonium* gives a black precipitate. 3. Tincture of galls, a blue black. Nitrate of baryta will show the presence of sulphuric acid. In *organic liquids* the salt may be obtained by dialysis in a state fit for testing. (See p. 154.)

Muriate of Iron—Tincture of Perchloride of Iron.—This is an acid solution of peroxide of iron with alcohol. It is of a deep red-brown color, and is much used in medicine. Dr. Christison relates an instance in which a man by mistake swallowed an ounce and a half of this liquid: the symptoms were somewhat like those produced by hydrochloric acid. He at first rallied, but died in about five weeks. The stomach was found partially inflamed, and thickened towards the lesser end. This salt has been much used for criminal purposes in France. (See *Medical Gazette*, vol. 47, p. 307; also *Ann. d'Hyg.*, 1850, vol. 1, pp. 180, 416; and 1851, vol. 1, p. 155, vol. 2, p. 337.) A case was reported to the Westminster Medical Society, in November, 1842, in which a girl, æt. 15, five months advanced in pregnancy, swallowed an ounce of the tincture of muriate of iron in four doses in one day, for the purpose of inducing abortion. Great irritation of the whole urinary system followed; but this was speedily removed, and she recovered. Another case of recovery from a large dose of this preparation has been reported by Mr. Amyot. A healthy married woman swallowed, by mistake for an aperient draught, *one ounce and a half* of the tincture of muriate of iron. She immediately ejected a portion, and violent retching continued for some time. There was great swelling of the glottis, with cough, and difficulty of swallowing. These symptoms were followed by heat and dryness of the throat, with a pricking sensation along the course of the gullet and stomach; and in the afternoon a quantity of dark grumous blood was vomited. The motions were black, owing doubtless to the action of sulphur upon the metal. In about a month the patient was perfectly restored to health. (*Provincial Journal*, April 7 and 21, 1847, p. 180.) Another case of recovery from a large dose has been reported by Sir James Murray. The patient, æt. 72, swallowed by mistake *three ounces* of the tincture in a concentrated state. The tongue soon became swollen; a ropy mucus flowed from the mouth and nose; there was croupy respiration, with a sense of impending suffocation. The pulse was feeble, the skin cold and clammy, and the face swollen and livid. A castor oil mixture brought away inky evacuations, and the patient rapidly recovered. (*Dub. Med. Press*, Feb. 21, 1849.) This liquid has been used in large doses for the purposes of criminal abortion. From the occurrence of these cases of recovery, it would be a mistake to infer that this was not a poisonous compound. The largeness of the dose has commonly led to early vomiting, and the rejection of the greater part of the acid liquid. Besides it varies much in strength, and unless this is known in any given case, it is difficult to draw a just inference from the quantity actually taken.

Comparatively small doses may seriously affect pregnant women, and among the criminal uses to which this preparation is put, may be mentioned that of procuring abortion. At the Lincoln Lent Assizes, 1863 (*Reg.* v. *Rumble*), a druggist was convicted of having supplied this noxious liquid to a woman with the intent to procure her miscarriage. He directed her to take a teaspoonful three times a day, and at the same time prescribed for her eight pills a day, each containing half a grain of powdered cantharides. Although the woman had taken only two doses of the tincture of perchloride of iron, she suffered from severe pain over the whole of the abdomen, with violent pain in the region of the stomach and bladder; there was constant vomiting of a greenish-colored matter, and great pain in passing her urine. The quantity of urine secreted was small, and contained much blood. These symptoms were in great part due to the cantharides. The proper dose of the iron-tincture is from ten to forty minims. Here it had been greatly exceeded, without any lawful excuse on the part of the prescriber. A case of recovery from an ounce of this tincture is quoted in the *Pharmaceutical Journal* (April, 1869, p. 605). A woman, æt. 30, swallowed this quantity. She suffered from vomiting and purging, the motions being black. Emetics were given, and she recovered in five hours. (*Lancet*, January 2, 1869, p. 9.)

The perchloride of iron has been used as an injection in uterine diseases; but it is a most powerful local irritant, and in one instance it caused death by inducing peritonitis. The symptoms were rigors, severe vomiting, and abdominal pain. The mucous membrane of the uterus was stained of a deep black, and iron was readily detected in its substance. (*Amer. Jour. Med. Sci.*, April, 1870, p. 566.)

Chemical Analysis.—The hydrochloric acid may be detected by nitrate of silver and nitric acid, while the peroxide of iron is immediately indicated by a precipitate of Prussian blue, on adding a solution of *Ferrocyanide of potassium.* The quantity of chloride present may be determined by evaporation.

COMPOUNDS OF BISMUTH.

Subnitrate of Bismuth—Pearl White—Magistery of Bismuth.—This substance, in a dose of *two drachms*, caused the death of an adult in nine days. There was burning pain in the throat, with vomiting and purging, coldness of the surface, and spasms of the arms and legs; also a strong metallic taste in the mouth. On inspection, the throat, larynx, and gullet were found inflamed; and there was inflammatory redness in the stomach and throughout the intestinal canal. (*Sobernheim*, p. 335.) In a case mentioned by the late Dr. Traill, a man took by mistake *six drachms* of the subnitrate in divided doses, in three days. He suffered from vomiting and pain in the abdomen and throat, but finally recovered. (*Outlines*, p. 115.) These cases are sufficient to prove that a substance very slightly soluble in water, may exert a powerfully poisonous action on the human system.

The oxide and subnitrate of bismuth, owing to imperfect washing, are frequently contaminated with arsenic in the form of arsenic acid. The symptoms produced by large doses have closely resembled those caused by arsenic, and as the medicinal subnitrate generally contains arsenic, the symptoms may have been on some occasions due to this impurity, as in the following cases. The first occurred to Dr. Fullertson, of Ohio. A physician in a neighboring state had occasion to place himself upon a treatment of subnitrate of bismuth. After a day or two he became aware of puffiness about his eyes and gastro-intestinal irritation. These symptoms soon became so pronounced that, knowing of no other possible cause for them than the bismuth, he discontinued its use, whereupon they subsided, but reappeared upon a renewal of the medicine. He then submitted a sample of the bismuth to a chemist, who, upon analysis, detected in it the presence of a formidable proportion of arsenic.

Dr. L. Hebert records (*Le Mouvement Medical*, Nov. 22, 1873) the case of an infant to whom the subnitrate of bismuth was given for a severe diarrhœa. Instead of being benefited by the remedy, the child presented symptoms of poisoning; on analysis the medicine was found to contain arsenic. (*Amer. Jour. Med. Sci.*, Jan. 1874.)

The ores of bismuth generally contain arsenic, and in preparing the subnitrate for medicinal use, sufficient care is not taken for removing the whole of the poison. I found arsenic in comparatively large proportion in samples obtained from three respectable retail druggists. Only two specimens out of five were free from this poison. The arsenic may be detected by dissolving the subnitrate in pure hydrochloric acid slightly diluted, and introducing it into Marsh's apparatus. The arsenical flame is apparent on combustion, and the usual deposits may be obtained on glass and porcelain. The products of combustion may be collected and tested by the processes described at p. 320. Bettendorff's test may also be applied to a suspected sample (see *ante*, p. 306). This impurity in the subnitrate may modify a conclusion respecting the presence of traces of arsenic in a body when bismuth has been administered medicinally. (See *Brit. and For. Med.-Chir. Rev.*, Oct. 1858.) A case in which a serious mistake was thus made (*State of Virginia* v. *Lloyd*, 1872), is recorded by Dr. Reese. On a charge of murder by poison, a fraction of a grain of arsenic was found in the liver of the deceased. In the defence it was attributed to impurity in the subnitrate of bismuth, which had been administered before death. The bismuth was examined, and found to contain arsenic. The prisoner was acquitted.

Analysis.—The subnitrate is a whitish chalky-looking uncrystalline powder, insoluble in water, dissolved by hydrochloric acid, and again precipitated white by dilution with water; the white precipitate is insoluble in tartaric acid, and is blackened by a solution of sulphuretted hydrogen, or by sulphide of ammonium. A solution of this substance in nitric acid gives no precipitate with diluted sulphuric acid.

COMPOUNDS OF CHROMIUM.

Bichromate of Potash—Symptoms and Appearances.—Well-observed instances of poisoning by this compound, which is now extensively used in the arts, are rare; and therefore the details of the following case, communicated to the *Medical Gazette*, vol. 33, p. 734, by Mr.

Wilson, of Leeds, are of practical interest. A man, aged sixty-four, was found dead in his bed twelve hours after he had gone to rest; he had been heard to snore loudly during the night, but this had occasioned no alarm to his relatives. When discovered, he was lying on his left side, his lower limbs being a little drawn up to his body; his countenance was pale, placid, and composed; eyes and mouth closed; pupils dilated; no discharge from any of the outlets of the body; no marks of vomiting or purging, nor any stain upon his hands or person, or upon the bed-linen or furniture. The surface was moderately warm. Some dyestuff, in the form of a black powder, was found in his pocket. On inspection, the brain and its membranes were healthy and natural; there was neither congestion nor effusion in any part. The thoracic viscera were equally healthy, as well as those of the abdomen, with the exception of the liver, which contained several hydatids. A pint of a turbid, inky-looking fluid was found in the stomach. The mucous membrane was red and very vascular, particularly at the union of the greater end with the gullet; this was ascribed to the known intemperate habits of the deceased. In the absence of any obvious cause for death, poison was suspected; and on analyzing the contents of the stomach they were found to contain bichromate of potash. The dye-powder taken from the man's pocket consisted of this salt mixed with cream of tartar and sand. It is worthy of remark that there was neither vomiting nor purging. The salt does not appear to have operated so much by its irritant properties as by its indirect effects on the nervous system. This, however, is by no means an unusual occurrence, even with irritants far more powerful than the bichromate of potash. A case has been communicated to me by Mr. Bishop, of Kirkstall, in which a boy recovered from the effects of a dose of this salt only after the lapse of four months. The first symptoms were pain, vomiting, dilated and fixed pupils, cramps in the legs, and insensibility. His recovery was due to early treatment. (See *Guy's Hosp. Reports*, Oct. 1850, p. 214.)

Another case, in which, owing to timely and proper treatment, a man, æt. 37, recovered from a large dose of this salt, has been communicated to me by Dr. H. C. Andrews (July, 1869). It seems that with suicidal intent, the man swallowed about two ounces of the bichromate in solution, mixed with pearlash. In about two hours he was seen by Dr. Andrews, and he was then apparently in a dying state. He was suffering chiefly from severe cramps, the pupils were dilated, the pulse was scarcely perceptible, and there was vomiting and purging of greenish-colored evacuations. The stomach-pump was used, and olive oil and diluents were given. In about nine hours the urgent symptoms abated, and the man complained only of great pain in the shoulders and legs. There was no gastric irritation nor tenderness of the abdomen. He was discharged cured at the end of a week. In a case which occurred to Dr. Schrader, a woman, æt. 24, died from the effects of this salt taken for the purpose of procuring abortion. The symptoms were those of an irritant—severe pain, vomiting, and purging. (Horn's *Vierteljahrsschrift*, 1866, vol. 2, p. 113.)

There can be no doubt that bichromate of potash is an active poison.

Mr. West has published a case from which it appears that a medical man, who had inadvertently tasted a solution of it, suffered from severe symptoms resembling those of Asiatic cholera. (*Provincial Jour.*, Dec. 24, 1851, p. 700.) Mr. Wood, of St. Bartholomew's Hospital, has furnished me with the particulars of a case in which two drachms of this substance destroyed the life of a woman in *four hours*. In the first two hours she suffered from violent vomiting and purging, the vomited matters being of a yellow color. When admitted she was in a dying state, pulseless, unconscious, and breathing slowly with great effort. The skin was cold; the lower lip swollen and purple, and the tongue swollen. The chief appearances were a dark and liquid state of the blood; the mucous membrane of the stomach was in great part destroyed, of a dark-brown color, approaching to purple; the duodenum at its upper part of a florid red color, and at its lower part much corrugated, as well as the upper half of the jejunum.

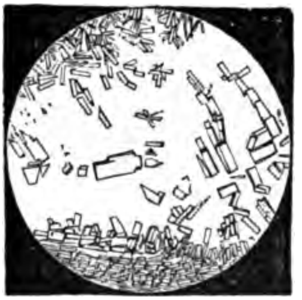
Dr. Baer, of Baltimore, has reported the following case. A man, in drawing off a solution of the bichromate by a siphon, accidentally received a small quantity into his mouth. In a few minutes he perceived great heat in the throat and stomach, and this was followed by violent vomiting of blood and mucus. The vomiting continued incessantly until his death, which took place in *five hours*. On dissection, the mucous membrane of the stomach, duodenum, and about one-fifth of the jejunum, was destroyed in patches. (Beck's *Med. Jur.*, p. 823.) In this case the salt acted as a corrosive irritant.

This salt, in a state of fine powder or in a saturated solution, has a local irritant action on the skin and on parts from which the skin has been removed. It produces sores, affecting chiefly the hands and exposed parts of the face. Thirty grains of this salt introduced into a wound in the back of a dog, produced vomiting, paralysis of the hind legs, and death in eleven hours. (*Brit. and For. Med. Rev.*, 1839, v. 14, p. 506.) The *chromate* of potash also has a poisonous action, but the green oxide of chromium is inert, according to Dr. Berndt (*loc. cit.*). Chromic acid is a powerful corrosive poison, destroying all organic textures. (Dr. Dougall, *Pharm. Jour.*, Jan. 1872, p. 568.)

Treatment.—Besides emetics, carbonate of magnesia or chalk, mixed up in a cream with milk, or albumen and water, should be given.

Analysis.—This is an acid salt, readily distinguished from all the other metallic poisons by its crystals having a deep orange-red color. They generally assume the shape of long four-sided tables, and sometimes a lengthened prismatic form. (See Fig. 38.)

FIG. 38.



Crystals of bichromate of potash, magnified 30 diameters. They have a deep orange-red color.

The salt is soluble in water, and the solution has a rich orange-red color. The solution has an acid reaction, and may be identified by the following tests: 1. Nitrate of silver gives a deep red precipitate. 2.

Acetate of lead gives a bright yellow precipitate. 3. Nitrate of baryta, a pale yellow; and, 4. Sulphuretted hydrogen gas gives a green precipitate. Boiled with alcohol and sulphuric acid it gives a green precipitate of oxide of chromium. From organic liquids it may be readily separated by dialysis. (See p. 154.)

CHROMATE OF LEAD.

This is a rich yellow or orange-colored compound, very much used as a pigment in the arts. Although seen in every color-shop, it is rare to hear of any accident arising from its use. It is said to have been used for coloring lozenges and other articles of confectionery, but if ill effects have followed, they have probably been set down to other causes.

Dr. Von Linstow, of Ratzeburg, has recently reported the cases of two children, under four years of age, in which this compound destroyed life. They had been playing with some substances made to look like bees, consisting of gum tragacanth, colored with chromate of lead. They ate a number of them. In two or three hours they were seized with violent vomiting and great prostration of strength. The matter at first thrown up had a yellowish color. The children were flushed in the face, complained of much thirst, and were very restless. There was no diarrhœa, and no complaint of pain. On the day following, the younger child had slight purging, with convulsions, and died on the second day. The elder was listless and almost unconscious; the face hot and flushed, the skin of the breast and abdomen erythematous; the pulse irregular; great thirst, with difficulty of swallowing. He passed into a state of collapse, and died on the fifth day.

On inspection of the younger child, the mucous membrane of the stomach was found thickened and swollen, with a number of red points scattered over it, some of them in groups. Near the cardia it had a pale-yellow color. The lungs and brain were much congested. In the elder child the appearances were similar.

On analysis no trace of chromate of lead or of lead could be detected. There were slight traces of copper, to which no importance could be attached. (Eulenberg, *Vierteljahrs.*, 1874, vol. 1, p. 607.) It is remarkable that no trace of lead was found in the tissues. The quantity of chromate taken could not have been large, and there had been much vomiting. Its action in some respects resembled that of a corrosive; it was wholly unlike that of a salt of lead.

Analysis.—This compound would be in general recognized by its brilliant yellow color, its complete insolubility in water, and its solubility in nitric acid, with the effect produced by sulphuretted hydrogen on the nitrate of lead formed.

COMPOUNDS OF THALLIUM.

The salts of this metal are, according to M. Paulet, highly poisonous, although this does not appear either from his own statement of their effects, or from the experiments of M. Lamy. M. Paulet found that a dose of fifteen and a half grains of carbonate of thallium killed a rabbit in a few hours. The animal suffered from disturbance of breathing, loss of muscular power, and general trembling of the limbs; it appeared to die asphyxiated.

Lamy dissolved seventy-five grains of the sulphate in milk, and he found that this quantity sufficed to destroy two hens, six ducks, two puppies, and a middle-sized bitch. The prominent symptoms in the dogs were oppression of breathing, salivation, griping pains in the abdomen, the body being drawn up, with trembling and convulsions of the limbs, followed by paralysis. Vomiting and purging are not described among the symptoms. Two of the puppies did not die until four days after they had taken the poison. On opening the bodies of the animals, Lamy states that there was no mark of inflammation or other striking post-mortem appearance. In one experiment he found that a puppy died in forty hours from a dose of one grain and a half of the sulphate of thallium. (*Chem. News*, Sept. 12 and 19, 1863.)

The salts are soluble, colorless, and nearly tasteless, and therefore may be easily administered. They have been found to operate through the skin and cellular membrane by absorption.

The statements of MM. Lamy and Paulet are not in accordance with the views of the discoverer of the metal, Dr. Crookes. Although much exposed to the action of the fumes, the metallic vapor produced no particular effects upon him. He also swallowed a grain or two of the salts without injury. These have a local action on the hair and skin, staining the former, and rendering the latter yellow and horny. (*Chem. News*, October 3, 1863, p. 161.)

Analysis.—According to these experimentalists, the best and most certain method of detecting thallium or its oxide or salts, if used as poisons, is to dry and burn the viscera, when, by the aid of spectrum-analysis, the green band indicative of thallium will manifest itself in the spectrum from the smallest quantity of the metal, in spite of admixture with other bodies.

The history of thallium as a poison is at present very incomplete. The above facts fail to show that it is an energetic substance.

SALTS OF PLATINUM, PALLADIUM, AND OTHER METALS.

The salts of *Platinum*, *Palladium*, *Iridium*, *Rhodium*, *Osmium*, *Cobalt*, *Nickel*, *Manganese*, *Cerium*, and *Uranium*, also possess an irritant action, partly, however, depending on the acids with which they are combined. They are products of art not met with in common life; and, so far as I can ascertain, they have never been taken as poisons by man. It is unnecessary, therefore, to occupy space by detailing the chemical processes whereby they may be identified; these will be found fully described in all works on chemistry.

Osmic Acid, from ὀσμή (odor).—This is a compound of the metal osmium with oxygen. Although called an acid, it does not redden litmus when dissolved in water. It has a remarkably pungent and acrid odor, resembling that of the chloride of sulphur. It has an acrid, burning taste, and in a vaporous state, which it easily assumes, it is most irritating to the eyes and lungs, exciting severe cough and expectoration, rendering irrespirable a large quantity of air. Its action is that of an irritant, and, like the vapor of nitric acid, if breathed, it would produce similar effects on the lungs, leading to death. Gmelin describes it as a poison to animals. Its effects on man are unknown. *Osmic acid* is a white, translucent, crystalline substance—soft, like wax, at a moderate heat. It melts, boils, and evaporates below 212°, and it may be obtained by condensation of the vapor in transparent crystalline prisms. It is slowly dissolved by water. The solution has a strong odor and taste, and stains the skin black.

COMPOUNDS OF TIN.

The only preparations of this metal which require to be noticed as poisons are the *chlorides* or *muriates*, a mixture of which is extensively used in the arts, under the name of *Dyer's spirit.* The salts may exist in the form of whitish-yellow crystals; but more commonly they are met with in a strongly acid solution in water. They are irritant poisons; but so seldom used as such that only one death occurred in England and Wales during a period of two years.

COMPOUNDS OF SILVER.

Nitrate of Silver—Lunar Caustic—Lapis Infernalis.—This substance, which is commonly met with in small sticks of a white or dark gray color, is readily soluble in distilled water; in common water it forms a milky solution. It acts locally as a powerful corrosive destroying all the organic tissues with which it comes in contact. There are several cases on record in which it has proved fatal in the human subject; one of these occurred in 1837–8, but the particulars are unknown. The symptoms come on immediately, and the whitish flaky matter vomited is rendered dark by exposure to light. The presence of dark-colored spots on the skin will also indicate the nature of the poison. In September, 1861, a woman, æt. 51, died in three days from the effects of taking a six-ounce mixture containing fifty grains of nitrate of silver (lunar caustic) given in divided doses. She vomited a brownish-yellow fluid before death. The stomach and intestines were found inflamed. It is stated that silver was found in the substance of the stomach and liver. A well-marked case of poisoning with this substance occurred to Mr. Scattergood. A portion of a stick of lunar caustic dropped down the throat of a child aged fifteen months. In spite of treatment the child died in six hours in violent convulsions. (*Brit. Med. Jour.*, May 27, 1871; and *Amer. Jour. Med. Sci.*, July, 1871, p. 287.) In the *treatment* of these cases a solution of chloride of sodium with albumen should be freely given.

COMPOUNDS OF GOLD.

Perchloride.—This is the only preparation of gold which requires notice. It is a powerful irritant poison, acting locally like the nitrate of silver. Nothing is known of its effects on the human subject; but in administering it to animals, Orfila found extensive inflammation, and even ulceration, of the mucous membrane of the stomach. (*Toxicologie*, vol. 2, p. 30.) The metal is absorbed and carried into the tissues, but its poisonous action appears to be wholly independent of absorption.

VEGETABLE IRRITANTS.

CHAPTER LI.

Action of vegetable irritants—Savin—Symptoms and appearances—Oil of savin—Its properties—Croton seeds and oil—Fatal effects—The physic-nut, or Jatropha curcas—Capsicum—Charlock, or wild mustard—Its irritant properties on cattle—Gelsemium semper virens—Yellow jasmine.

The poisonous substances of an irritant nature which belong to the vegetable kingdom are very numerous as a class; but it will here be necessary to notice only those which have either caused death or given rise to accidental poisoning. The true *vegetable* irritants, soon after they are swallowed, produce severe pain in the abdomen, accompanied by vomiting and purging. There are rarely any cerebral symptoms, and no convulsions.

It must be admitted, however, that the operation of many of them is by no means clearly defined. Stupor, delirium, and convulsions have been occasionally observed as secondary effects; hence the distinction between some vegetable irritants and those which are assigned to the neurotic class is purely arbitrary. Further experience may hereafter lead to a better knowledge of their *modus operandi*, and to an improved classification. One circumstance is worthy of remark. The effects of neurotic poisons can commonly be traced to the presence of a poisonous alkaloid in the vegetable. Among the irritants, the effects appear to be frequently due to the presence of an acrid oil or resin.

Some of the vegetable irritants act especially on the bowels, and, in mild doses, are safely used as purgatives. In large doses they produce violent purging, and in old and young persons are apt to cause death by exhaustion. There are, however, but few instances recorded of their fatal action on the human body; and the little that is known concerning their operation as poisons, is chiefly derived from experiments performed on animals. The changes found after death are confined to irritation and inflammation of the alimentary canal. These substances (if we except savin) are rarely resorted to by the suicide or murderer, for large doses are required, and their fatal operation even in these cases is rendered uncertain by the circumstance that they excite vomiting, and are then commonly expelled from the stomach.

Treatment.—In cases of poisoning by the vegetable irritants, emetics and purgatives (castor oil) or injections should be freely employed, and when the poisonous vegetable is expelled, antiphlogistic measures may be used.

SAVIN—JUNIPERUS SABINA.

This is a well-known plant, the leaves or tops of which contain an irritant poison in the form of an acrid volatile oil of a peculiar terebinthinate odor. They exert an irritant action, both in the state of infusion and powder. They yield by distillation about three per cent. by weight of a light-yellow oil, on which the irritant properties of the plant depend. The powder is sometimes used in medicine in a dose of from five to twenty grains. Savin is not often taken as a poison for the specific purpose of destroying life; but this is occasionally an indirect result of its use, as a popular means for procuring abortion, and it therefore demands the attention of a medical jurist. From cases which have been referred to me, I believe that poisoning by it is more frequent than is commonly supposed.

Symptoms and Appearances.—From the little that is known of its effects, savin acts by producing violent pain in the abdomen, vomiting, and strangury. Purging is not so common an effect as with other irritants. Salivation is sometimes present. After death, the gullet, stomach, and intestines, as well as the kidneys, have been found either much inflamed or highly congested. There is no proof of its having any action as an abortive, except, like other irritants, by causing a violent shock to the system, under which the uterus may expel its contents. Such a result can never be obtained without placing in jeopardy the life of a woman; and when abortion follows, she generally falls a victim. On the other hand, a woman may be killed by the poison without abortion ensuing. Out of four fatal cases of the administration of savin and other drugs for the purpose of procuring abortion, the mother died undelivered in three, and in the fourth instance, the child died soon after it was born. When the vomiting and purging are very severe, abortion may be expected to follow.

The strong local irritant properties of the leaves, which depend on the essential oil, are well known, from the uses of savin-ointment in pharmacy. The plant grows extensively in country places, and is easily accessible to the evil-disposed. It does not appear to have attracted much notice on the continent, for Orfila is silent on poisoning by this substance, except in so far as it affects dogs. Two cases of its fatal effects in the human female were communicated to Sir R. Christison. In one, a dose of the strong infusion was twice taken by a woman for inducing abortion. She suffered from severe pain and strangury, aborted, and died five days afterwards. On inspection there was extensive peritoneal inflammation, with the effusion of fibrinous flakes; the inside of the stomach was red, with patches of florid extravasation. The contents had a green color, and savin was proved to be present by the aid of the microscope. In the second case a girl was seized with violent colicky pains, vomiting, tenesmus, difficulty in passing urine, and fever. After suffering several days, she died. The stomach and intestines were inflamed; the former was in some parts black, and at the lower curvature, perforated. A greenish powder was also found in this case, and when washed and dried, it had the pungent odor and taste of savin.

Although it is not considered that savin has a direct tendency to produce abortion, it appears, from its therapeutic employment in chlorosis and amenorrhœa, to affect the uterus. The dried powder, which, owing to the loss of volatile oil, is less energetic than the fresh tops, is given in doses of from five to fifteen grains. The medicinal dose of the essential oil is commonly from two to six drops. The infusion and decoction, which are sometimes used for the expulsion of worms, are less energetic than the fresh tops, because they cannot be prepared without giving rise to a loss of the volatile oil. The oil is not so irritant as it is commonly supposed to be; but in those cases in which it has been said to produce no injurious effects in large doses, it is probable that it was much adulterated.

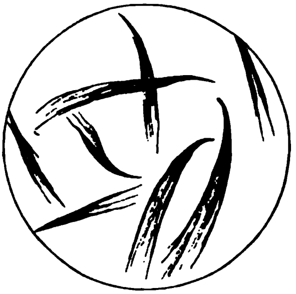
A well-marked case of poisoning by the tops of savin was referred to me in May, 1845. The deceased, a healthy woman, had reached about the seventh month of her pregnancy. She was very well on the Friday, but was seized with vomiting on the Saturday; she stated that she had taken nothing to produce it. The vomiting continued throughout Sunday, and was of a green color. She was first seen by a medical man on Sunday evening. The symptoms were those of inflammation of the stomach and bowels, with great anxiety, and the pulse 150. The green color of the vomited matter was at first supposed to be owing to altered bile. The vomiting appears to have continued at intervals, but it does not seem that there was any violent purging. Labor came on on Wednesday. The child was born living, but soon died; the woman herself died on the Thursday, *i. e.*, five days after having taken the poison, for there was no proof that any savin could have been taken after Saturday. On inspection, the brain was healthy, the lungs were healthy, except that the air-tubes had a dark red color, the heart was flabby, and the blood was generally fluid. The lining membrane of the gullet was reddened, and had on it ecchymosed patches. One-half of the mucous membrane, from the cardiac orifice upwards, presented a dark-red arborescent injection, with slight patches of ecchymosis; there was no erosion or ulceration. In the stomach a large patch of redness, about three inches in length, extended from the greater curvature towards the pylorus. The vessels of the mucous membrane were considerably injected, forming infiltrated patches, especially about the lesser curvature, extending towards the cardiac end; but there was no ulceration or erosion. The stomach contained nearly eight ounces of a greenish fluid, of the appearance and consistency of green-pea soup. By examining a portion of the washed vegetable substance under a microscope, and by drying a portion, rubbing it, and observing the odor, clear evidence was obtained that the green color was owing to the diffusion of finely triturated savin-powder. The interior of the duodenum, especially towards the pylorus, was intensely inflamed, being of the color of cinnabar. Patches of inflammation were found throughout the other portions of the intestinal canal. There was some inflammation of the peritoneum, chiefly of the upper part of the intestines and omentum. The kidneys were inflamed, and of a dark red color; the bladder was healthy. A green-colored mucous matter, containing savin, was found in the duodenum, but not in the lower part of the intestines. (*Med. Gaz.*, vol. 36, p. 646.) The quantity of poison taken by the deceased could not be ascertained, but it must have been large. I estimated the quantity remaining in the stomach after five days, under frequent vomiting, at from twenty-five to thirty grains.

In a case which occurred to Mr. Newth, the patient, a pregnant woman, eight hours after she had taken savin, was found lying on her back perfectly insensible, and breathing stertorously. She had been suddenly seized with vomiting, and this continued for some time. At first, the case was thought to be one of puerperal convulsions. Labor came on, and she died in about four hours, during a fit of pain. She appeared to be between the seventh and eighth month of pregnancy, and the child was born dead. On inspection, twenty-four hours after death, the brain was found gorged with black fluid blood. The stomach was paler than usual, excepting in one or two spots, which were red, as if blood had been effused into the mucous tissue. It contained four ounces of an acid liquid of a brownish-green color. This, on distillation, yielded an opaque liquid, from which a few drops of a yellow oil were separated by means of ether. Some sediment found in a bottle presented, under the microscope, the characters of powdered savin-leaves. (*Lancet*, June 14, 1845, p. 677.) There can be no doubt that this was the cause of death. The action of the poison appears to have been, in the first instance, like that of an irritant, and just before death like that of a narcotic.

Analysis.—When savin has been taken in the form of decoction or infusion, it is beyond the reach of chemical tests. The fact of poisoning can then only be elucidated by the symptoms and by circumstantial evidence. If the oil has been taken, it may be separated by distillation, and obtained by agitating the distilled product with one-third of its bulk of ether. Perhaps the most common case is that where the *powder* of the leaves has been taken. In two of the cases above related, it will be observed that, in spite of great vomiting, some of the powder remained in the stomach for a period of five days. The contents have generally the appearance of green-pea soup. That the color is not owing to bile may be proved by diluting a portion with water, when the green chlorophyll, owing to its insolubility, will subside in a dense stratum, whereas if the color were due to altered bile, the whole of the liquid would remain colored. By washing the green deposit in water, and drying it on a glass-slide or mica, evidence may be obtained under a good microscope, by the rectilinear course of the fibres and the turpentine-cells, that the substance belongs to the coniferæ. The only other poison of the coniferous order is the yew (*Taxus baccata*), but the leaf of this tree differs from that of savin in having a lancet-shaped apex, while savin has a sharply acuminated point. (Fig. 39.) A portion of the green powder dried and well rubbed will give the peculiar odor of savin. When freed from organic matter, it yields, by distillation with water, the essential oil of savin.

Oil of Savin.—This oil is of a light-yellow color, and it has a powerful terebinthinate odor, sufficiently peculiar to render this an easy means of identification. A greasy stain made by this oil on paper is entirely dissipated by heat, or only a slight trace of resin is left. It is lighter than water, but insoluble in it, giving to it, however, its odor and an acid reaction. It forms a milky solution with rectified spirit, but a clear transparent solution with ether. It is very soluble in ether, and by this menstruum it may be separated from watery liquids, as the ether floats with it to the top. The oil may be there obtained by allowing the ether to evaporate. Oil of savin must be regarded as a noxious substance, especially when given in large doses to pregnant women. It has been occasionally employed for procuring abortion. In *Reg.* v. *Pascoe* (Cornwall Lent Assizes, 1852) a medical man was convicted and sentenced to transportation for administering oil of savin to a woman with intent to procure miscarriage. The proof of intent rested partly on medical and partly on moral circumstances. It appeared that the prisoner had given fourteen drops of the oil, divided into three doses daily—a quantity which, according to the medical evidence at the trial, was greater than should have been prescribed for any lawful purpose. The medicinal dose, as an emmenagogue, on the authority of Christison, is from two to five *minims*, and according to Pereira from two to six *drops*. The quantity given by the prisoner, although a full dose, was not, therefore, greater than these authorities recommend; and his criminality appears to have rested not so much on the dose given, as on the question whether he knew, or, as a medical man, had reason to *suspect* that the female for whom he prescribed it, was pregnant. No medical authority would recommend oil of savin in full doses as a safe medicine for *pregnant* women; and with regard to the existence or non-existence of pregnancy in a special case, medical men are reasonably presumed to have better means of satisfying themselves than non-professional persons. The prisoner's innocence, therefore, rested on the presumption that he implicitly believed what the prosecutrix told him regarding her condition; that he had no reason to *suspect* her pregnancy, and therefore did not hesitate to select and prescribe a medicine which certainly has an evil reputation, and is rarely used by regular practitioners. According to the evidence of the prosecutrix, she informed the prisoner that she had disease of the heart and liver, and that nothing more was the matter with her. It is absurd to suppose that oil of savin would be prescribed by a medical man for such a disease as this. The prisoner, on the hypothesis of innocence, must have intended that the medicine should act on the uterus, and must have inferred the existence of an obstruction to menstruation from natural causes irrespective of pregnancy. The jury do

FIG. 39.



Tips of the leaves of savin, magnified 30 diameters.

not appear to have given him credit for such ignorance of his profession, and this probably led to his conviction.

There can, it appears to me, be no doubt that the oil was, in this case, administered with a guilty intention. Every qualified practitioner, acting *bonâ fide*, would undoubtedly satisfy himself that a young woman whose menses were obstructed, was *not pregnant*, before he prescribed full doses of this oil three times a day, or he would fairly lay himself open to a suspicion of criminality. If pregnancy—a frequent cause of obstructed menstruation—were only *suspected*, this would be sufficient to deter a practitioner of common prudence from prescribing, in any dose, a drug which may exert a serious action on the uterine system. (A report of the case of Mr. Pascoe will be found in the *Med. Times and Gazette*, April 17, 1852, p. 104.) On the Northern Circuit, December, 1853 (*Reg.* v. *Moore*), a man was tried and convicted of administering oil of savin to a pregnant woman. It made her very ill, but did not produce abortion.

Analysis.—In cases in which this oil has been given, and has operated fatally, it may be separated from the contents of the stomach by ether. The odor of the oil is stated to have been perceived after death in the blood and in the cavities of the body. This may be regarded as the best test of its presence. (See a paper by Dr. Lex, in Horn's *Vierteljahrsschrift*, 1866, pp. 1, 241.) The oil of savin forms a turbid mixture with alcohol (.826). When treated with its volume of sulphuric acid, it acquires a dark-brown color, and when this mixture is added to distilled water, a dense white precipitate is separated.

CROTON SEEDS AND OIL (CROTON TIGLIUM).

This is a fixed oil extracted by pressure from the seeds of the Croton tiglium. The seeds, which are sometimes called *Purging nuts*, resemble castor seeds in size and shape. (See Fig. 40, p. 485.) They have no smell. Their taste is at first mild and oleaginous, afterwards acrid and burning. When heated they evolve an acrid vapor. Croton oil is a powerful drastic purgative, producing, in a large dose, severe purging, collapse, and death.

Symptoms and Appearances.—The seeds owe their poisonous properties to the presence of this oil. One or two grains of the *seeds*, when swallowed, are sufficient to produce severe pain in the abdomen, with copious watery motions. Even the dust of the seeds, when inhaled, has caused alarming symptoms. Dr. Pereira mentions the following case: A man had been occupied eight hours in emptying packages of the seeds, and had thus been exposed to the dust. He first experienced a burning sensation in the nose and mouth, tightness in the chest, effusion of tears, and pain in the pit of the stomach. He then became giddy, and fell down insensible. When admitted into the hospital the man was in a state of collapse, complained of burning heat in the stomach, throat, and head, and of swelling and numbness of the tongue. The region of the stomach felt hot and tense, the pupils were dilated, the breathing short and hurried, pulse 85, and the skin was cold. He had pain in the epigastrium for several days; but it is singular that there was no purging. (*Mat. Med.*, vol. 2, pt. 1, p. 406.) In March, 1874, various articles were washed ashore in Waterford Harbor from the wreck of a vessel which had foundered at sea. A large quantity of foreign nuts resembling small beans were picked up and eaten by the country people. Twenty-four persons ate them. They were all attacked with symptoms of irritant poisoning, but recovered. The seeds were proved to be those of the croton tiglium. (*Med. Times and Gaz.*, 1874, p. 272.)

The *oil* has a hot burning taste. One or two drops are commonly sufficient to produce pain in the abdomen, and purging; but Dr. Traill states that a woman usually took three drops for a dose without inconvenience—an effect of habit. (*Outlines*, p. 149.) In one case a teaspoonful was given, by mistake, to a child æt. 4, who had previously eaten a full meal of bread and milk. In five minutes, the child was seized with violent vomiting and purging, and these symptoms were soon followed by alarming prostration. Under the use of warm fomentations, and of milk and mucilage, the child recovered in two days. (Dr. Cowan in *Prov. Trans.*, N. S., vol. 1, p. 121.) The recovery was here probably due to the oil having been taken on a full stomach and to early vomiting. Dr. Cowan states that he has known similar symptoms to have been produced in an adult by *half a drop* of the oil. In large doses, the pain is described as hot and burning, extending from the mouth downwards; there is violent vomiting with purging, and the patient rapidly sinks exhausted. After death, the alimentary canal is found inflamed. Even the endermic application of the oil is stated in some cases to have produced severe symptoms, although, according to Dr. Buchanan, it acts only as a local irritant. (*Medical Gazette*, vol. 39, p. 671.)

A case occurred in Paris in 1839, in which a man swallowed by mistake two drachms and a half of croton oil. In three-quarters of an hour the surface was cold and clammy, the pulse imperceptible, respiration difficult, and the extremities and face were as blue as in the collapsed stage of cholera. In an hour and a half purging set in; the stools were passed involuntarily, and the abdomen was very sensitive to the touch. The patient complained of a burning pain in the course of the œsophagus. He died in four hours after swallowing the poison; and it is singular that there was no marked change in the mucous membrane of the stomach. (Orfila, *Tox.*, vol. 1, p. 108.) In June, 1855, a patient in the Dumfries Infirmary swallowed by mistake about three drachms and a half of a liniment containing croton oil. In a few minutes he experienced a violent burning sensation, extending from the throat to the stomach, with severe pain in the stomach. He complained of a spasmodic suffocative feeling, and convulsively gasped for breath for several minutes. At first he felt strongly inclined to vomit, but was quite unable to do so. He was then attacked with severe vomiting and purging. An emetic was given, and vomiting was kept up by various means for nearly an hour. At the end of this time the man became faint; the skin was cold and pale; and the face and lips assumed a livid tint. The pulse was small, and almost imperceptible; and he was unable any longer to maintain the erect or sitting posture.

Under treatment, the local pain and general distress was much decreased; the vomiting was arrested; and the surface of the body gradually regained warmth. On the fourth day the mucous membrane of the tongue and throat came away in shreds; and the uneasiness of the gullet was diminished. On the sixth day the patient had quite recovered, but felt rather weak. (Case by Dr. Adam, *Ed. Med. Jour.*, 1855-6, vol. 1, p. 932.)

In the *Medical Gazette* there is a report of a case in which a woman died from the effects of an embrocation containing croton oil, with other drugs. A teaspoonful was incautiously given to her; she immediately complained of a hot burning sensation in her throat. She was an aged person, and died in convulsions in three days. (*Med. Gaz.*, vol. 43, p. 41.) A girl, æt. 19, took by mistake a teaspoonful of a liniment consisting of equal parts of croton and olive oil. In about half an hour she was seen by Dr. Brydon, and she then complained of an intense burning sensation in the throat and gullet; but there was no pain in the stomach. Her pulse was 84. Vomiting came on in a severe form, and this was promoted by a zinc emetic and warm water. After the vomiting had continued for a quarter of an hour, she complained of a severe pain in the stomach. Purging was not a prominent symptom. In a day or two she recovered. (*Edinburgh Medical Journal*, Aug. 1861.) In another case a little girl, six years old, took by mistake about fifty-five drops of croton oil. There was vomiting, with some purging and feverishness for three or four days, but the patient recovered. (*Lancet*, 1870, vol. 1, p. 553.) In these cases it is not improbable that the oil may have been weakened by adulteration. In one instance reported, a child, æt. thirteen months, died in six hours from a small dose given by mistake. The croton oil was mixed with soap liniment, and the quantity taken was supposed to be less than three minims of the oil. (*Med. Times and Gaz.*, 1870, vol. 2, p. 466.)

M. Chevallier reports two cases of poisoning with this oil. In one a druggist swallowed by mistake for cod-liver oil half an ounce of croton oil. He felt a burning sensation in his throat and stomach, and this was soon followed by vomiting and copious purging, with symptoms of collapse. He did not recover until after a fortnight. In the other case, quoted from Devergie, a man, æt. 25, swallowed by mistake two drachms and a half of the oil. The most violent purging with collapse took place, and the patient died in four hours. (*Ann d'Hyg.*, 1871, vol. 1, p. 409.)

A case was tried at the Liverpool Winter Assizes (*Reg.* v. *Massey and Ferrand*), in which the prisoners were charged with having caused the death of a man by placing in food, of which he and others had partaken, two drachms of powdered jalap and from two to six drops of croton oil. Several persons, including the deceased, suffered from vomiting and purging; but they recovered, and the deceased himself so far recovered as to be able to go about as usual. He was subsequently attacked with inflammation and ulceration of the bowels, from which he died. The prisoners were acquitted, as the medical evidence failed to make out a direct connection of this secondary illness with the jalap and croton oil which had been put into the food. Dr. Ellis has reported a case which was the subject of a recent trial for murder in the United States. The prisoners were charged with having caused the death of a man, æt. 35, by giving him two drachms of croton oil in a glass of whisky. It was proved that the oil had been given to deceased, an habitual drunkard, when he was intoxicated, at 9 P.M. He vomited, but was not purged, and was found dead the next morning. On inspection the heart, lungs, and brain showed nothing unusual. The mucous membrane of the stomach and small intestines was much inflamed and in some parts absorbed. Mixed with the chyme in the stomach there was a film of oil, having the peculiar smell of croton oil; it was separated as *cròton oil* by an alcoholic solution of soda and the subsequent addition of hydrochloric acid. It had the acrid properties of croton oil. (*Amer. Jour. Med. Sci.*, April, 1874, p. 416.) The symptoms and appearances, however, left it doubtful whether they had really been caused by the oil. Violent purging is the most striking symptom, but this was absent. The condition of the mucous membrane of the stomach and intestines was such as might have been produced by excessive drinking—alcoholism. There could be no doubt that the oil was given, but there was no conclusive evidence that it was the cause of death.

The poisonous qualities of croton oil are owing to a fatty acid (crotonic acid), which it contains in uncertain quantity. Probably this may explain why from six to ten drops of the oil may be sometimes given without causing much purging. It commonly begins to act speedily, *i. e.*, within half an hour. The medicinal dose of it is from one to three drops. The oil acts as a poison on animals. Many instances of its action on animals have been collected by Wibmer. (*Arzneimittel*, vol. 1, p. 215.) A singular case in reference to its effects on the horse, was the subject of a trial some years since. A veterinary surgeon administered, as a medicine, fifteen drops to a horse. The lips of the animal were swollen, and the skin peeled off; the horse suffered evidently great pain, and after lingering a short time, died. An action was brought by the owner of the horse at the Oxford Aut. Cir., 1838, for the recovery of its value. From the evidence then given, it seemed probable that the animal had really died from a small dose of the oil, although there was reason to believe that a larger quantity was given than was here alleged to have caused death. Wibmer mentions two instances in which twenty and thirty drops were given to horses without materially affecting them.

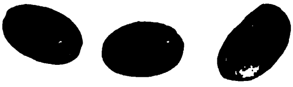
Fatal Dose.—In man, a dose of from fifteen to twenty drops of the *pure* oil might give rise to excessive purging, and cause death by exhaustion. The cases recorded of its fatal operation are few, and do not enable us to solve this question from observed facts. According to Landsberg, as quoted by Christison (*Dispensatory*, p. 382), thirty drops of the oil killed a dog; and Sir R. Christison states that he has known four grains of the oil to produce an alarming degree of purging. It is frequently mixed with castor oil and other substances, and the presence of these must of course influence the dose required to act fatally.

Treatment.—The seeds or oil should be removed from the stomach by emetics or the stomach-pump. Demulcent drinks may be freely given.

Analysis.—The croton oil of the shops is a brownish or yellow-colored fixed oil, of a peculiarly unpleasant odor, and a hot acrid taste. Rubbed on the skin, it produces, after a time, redness and a pustular eruption. It has an acid reaction, which it imparts to water, and, as it is of lower specific gravity, it floats on the surface. It is very soluble in ether, and by this means it may be separated from organic liquids. Ether may be employed for separating it from the contents of the stomach, and this liquid may be afterwards removed by evaporation or distillation. Croton oil is employed as a purgative in medicinal doses of from one-third to one minim; and also externally as a liniment, which contains one-eighth of its volume of the oil. There is nothing characteristic in reference to its chemical properties. When warmed with nitric acid, the oil acquires a dark-brown color, and there is an abundant evolution of nitrous acid vapor.

Croton *seeds* are of an oval form, and about three-eighths of an inch in length. They are covered with a dusky thin bluish-colored brittle shell, having within a yellowish-white oleaginous and easily sectile kernel, which forms the great bulk of the seed. When boiled in a solution of potash holding dissolved some oxide of lead, they are blackened, thus indicating the presence of sulphur. Like all the varieties of vegeto-albumen, the kernel is turned of a deep red-brown color when it is boiled in concentrated hydrochloric acid.

FIG. 40.



Seeds of croton tiglium, natural size.

PHYSIC NUT (JATROPHA CURCAS).

The Jatropha Curcas is a West Indian plant, which produces seeds containing an acrid oil, having some of the properties of croton oil. Four seeds act as a violent cathartic, and severe vomiting and purging have been produced by a few grains of the cake left after the expression of the fixed oil from the bruised seeds. The oil operates powerfully in a dose of from twelve to fifteen drops. It produces a burning sensation in the throat, vomiting and purging, and other symptoms of irritation, followed by inflammation of the stomach and bowels. In August, 1858, 139 children in Dublin suffered from the effects of these seeds or nuts (*Med. Times and Gaz.*, Aug., 1858); and in June, 1864, a number of boys at Birmingham suffered severely from eating some of them which they had found in a drug-store, but they all recovered. M. Chevallier refers to a case in which thirty-three persons were poisoned by eating the seeds. The *symptoms* from which they suffered were nausea, vomiting, and general depression. Twenty were so ill that they were placed in the beds of a hospital; the remaining thirteen soon recovered. The albumen of this seed, as well as that of the castor, is said to have a flavor resembling that of the almond. (*Annales d'Hyg.*, 1871, vol. 1, p. 408.)

The *Jatropha urens*, also a West Indian plant, is said to produce serious effects upon those who touch its leaves, which are covered with stinging hairs like those of the nettle. One of these plants was raised at Kew from seeds sent from Trinidad. Mr. Jackson, of the Kew Museum, gives the following account of the effects of contact. The wrist of a person accidentally came in contact with some of the hairs. In a few minutes there was swelling of the lips, redness of the face, faintness, great prostration of strength, and such a degree of collapse, that for some minutes the sufferer was thought to be dead. He then rallied; there was sickness, and in twenty minutes the man recovered. In another case the pain and swelling in the part touched, lasted for some days, and an itching sensation continued for a longer period. (*Pharm. Jour.*, April 17, 1872, p. 863.) Assuming this account of the symptoms to be correct, the poison connected with the hairs not only has a local action, but it is very rapidly absorbed, and produces effects resembling those of serpent-poison.

CAYENNE PEPPER—CAPSICUM.

A trial for manslaughter, which took place at the Central Criminal Court (*Reg.* v. *Stevens*, May, 1864), renders it necessary to notice a substance much better known as a condiment than as a vegetable irritant poison. In this case a medical botanist was charged with having caused the death of the deceased, a boy, æt. 15, by administering to him dangerous medicines. The boy was suffering from diseased hip-joint, and, after taking the medicines prescribed by the prisoner, he died. Dr. Letheby examined the stomach, and found in it patches of inflammatory redness, such as would be produced by an irritant. He could detect no poison, but simply bilious matter mixed with cayenne pepper. The mixture prescribed by the prisoner contained this pepper, which the witness considered to be injurious to a person in the condition of deceased. The prisoner was acquitted, the connection of the death of deceased with his act being probably considered by the jury as not proved.

In small quantities, this is a well-known stimulant and a useful condiment and medicine. It has a hot, fiery taste, which lasts for a long time on the parts which it touches. It acts as an irritant, and in large doses produces difficulty of swallowing, pain in the stomach, and inflammation of the gullet and stomach. Locally applied, it causes redness and even blistering of the skin. (Wibmer, *Arzneimittel*, art. "Capsicum.") There is no instance recorded of its having proved fatal. It owes its irritant properties to an acrid resin (*capsicin*), of which it contains four per cent. From five to ten grains of the powder are considered to be a medicinal dose.

Guinea Pepper, known as Grains of Paradise, is popularly considered to be highly noxious; but there are no facts to justify this view. This kind of pepper is properly regarded as an aromatic condiment.

CHARLOCK OR WILD MUSTARD (SINAPIS ARVENSIS).

The seeds of this plant, as well as those of the black mustard (*Sinapis nigra*), have been long known to possess irritant properties. When ground to the state of flour, and mixed with tepid water, an acrid pungent volatile oil is produced. By contact with the skin this substance produces irritative inflammation, and, when carried further, even a sloughing of the soft parts.

The flour of mustard (*Sinapis nigra*) deprived of its fixed oil, is used as a condiment. It possesses a hot pungent taste and odor, and, when taken into the stomach, produces vomiting. It is usefully employed as a good domestic emetic in most cases of poisoning, in the dose of a dessertspoonful to three or four ounces of tepid water.

When taken in large quantity, and not speedily ejected, it causes inflammation of the stomach and bowels, and thus operates as an irritant poison. Such cases are not met with among human beings; but, in veterinary practice, it is well known that this is a not unfrequent cause of death among cattle fed on oil-cake, especially that derived from the compression of rape-seed in the manufacture of colza oil.

The rape-cake is not in itself injurious; but the seed is very frequently mixed with that of the charlock (*Sinapis arvensis*). The bruised seeds are thus compressed into one hard, dark-colored mass. When eaten by the cattle, the fluids of the body are sufficient to produce the irritating essential oil, and the animal dies from gastritis or gastro-enteritis. In December, 1874, Professor Voelcker informed me that a set of cases had been referred to him in which eight bullocks had died from this cause.

Actions are occasionally tried in our courts involving this question. Damages are claimed for the loss of cattle by the supply of improper food.

Analysis.—The process for detecting the presence of mustard is simple. A small quantity of the powdered cake, ground seeds or flour, is mixed into a paste with tepid water, and allowed to stand for a short time. The pungent taste and smell acquired will indicate the presence of the essential oil of mustard. A decoction of the seeds, in some respects, resembles a solution of opium. It strikes a red color with nitric acid and a persalt of iron, and it decomposes iodic acid, setting free iodine. The red color produced by a persalt of iron is destroyed by a few drops of dilute acid. It is, in fact, sulphocyanide of iron, and differs from the meconate of iron. (See Meconic Acid, *post.*)

YELLOW JASMINE (GELSEMIUM SEMPERVIRENS).

An alcoholic extract of the root of this plant has been used in the United States for medicinal purposes. It has acted as a poison and destroyed life, but its exact place as a poison cannot yet be satisfactorily assigned. From a case reported by Dr. Wormley (*American Journal of Pharmacy*, Jan. 1870), it appears to belong rather to the irritant than the narcotic class of substances.

A young healthy married woman took by mistake three teaspoonfuls of the fluid extract of gelsemium—a concentrated tincture of the root containing 480 grains to the ounce. She was several weeks advanced in pregnancy. In two hours after taking the extract she complained of pains in the stomach, nausea, and dimness of vision. These *symptoms* were followed by great restlessness, ineffectual efforts to vomit, and general perspiration. In four hours the pulse was feeble, irregular, and intermittent. There was great prostration, with irregular and slow breathing. The skin was dry, the limbs were cold, the pupils dilated and insensible to light; the eyes were fixed, and there was inability to raise the eyelids. The vital powers rapidly gave way, and, without convulsions, death occurred in seven hours and a half after the poison had been taken. On inspection the membranes and substance of the brain and spinal marrow were normal. The adipose tissue was thick and tinged with bilious matter. The lungs were collapsed, but natural in appearance, and the superficial veins were congested. The heart was normal—the veins on its surface were injected, and its cavities were distended with dark grumous blood, on the inside of which was a well-defined fibrinous deposit. The stomach contained a small quantity of ingesta; the peritoneum and intestines were in a healthy state. The left kidney was congested.

It will be seen from this account that, while death took place rapidly, there was nothing characteristic in the symptoms and appearances.

Analysis.—Dr. Wormley discovered that the extract contained an alkaloid (*gelseminin*) separable by ether or chloroform and an organic acid (*gelseminic acid*). The latter he was able to obtain crystallized in various forms by solution and sublimation. He found that if a small quantity of this acid or its salts in a solid state was treated with a drop of concentrated nitric acid, it became yellow or reddish, according to the quantity. When an excess of ammonia was added, it acquired a blood-red color. The hundredth part of a grain was sufficient for this reaction. The solution in potash is fluorescent, presenting a blue coloration on the surface. Gelseminic acid was thus detected in the contents of a stomach some months after death.

The alkaloid *gelsemin* or *gelseminin* is, according to Dr. Wormley, a potent poison. One-eighth of a grain by hypodermic injection killed a rabbit in one hour and a half. In fifteen minutes there were symptoms of great distress, and the animal was restless. In forty minutes there was great prostration, inability to move, respiration gasping, and the pupils were dilated, but there were no convulsions. From his experiments Dr. Wormley infers that the quantity which proved fatal to the woman in the abovementioned case could not have exceeded the sixth part of a grain! (*Amer. Jour. Med. Sci.*, April, 1870.)

CHAPTER LII.

CASTOR SEEDS: FATAL EFFECTS—POISONING WITH COLCHICUM—SEEDS, TINCTURE, AND ROOT—SYMPTOMS AND APPEARANCES—COLCHICINA—HELLEBORE AND ITS VARIETIES—VERATRIA: ITS EFFECTS—ERGOT OF RYE—SYMPTOMS CAUSED BY IT—ALOES, GAMBOGE, AND OTHER IRRITANTS.

CASTOR SEEDS (RICINUS COMMUNIS).

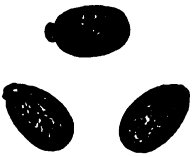
OF castor oil itself nothing need be said. It is not commonly known that the seeds, from which this oil is extracted, contain in the embryo an active poison, and that a few of them are sufficient to produce serious symptoms. Thus three or four seeds may act powerfully on an adult. Eight may give rise to serious symptoms, and a larger number may destroy life.

Symptoms and Appearances.—The symptoms which mark this form of poisoning, are the absence of a disagreeable taste or sense of heat in the mouth and throat at the time of eating the seeds. Soon after the pulp has been swallowed, there is severe pain in the abdomen, copious and painful vomiting, with bloody purging, thirst, and convulsions, terminated by death. (Bouchardat, *Ann. Thérapeutique*, 1872, p. 103.) A girl, æt. 18, the sister of a gentleman who was at the time attending my lectures at Guy's Hospital, ate about twenty castor-oil seeds; one of her sisters ate four or five, and another two. This was in the evening. During the night they were all taken ill. About five hours after the seeds were eaten, the deceased felt faint and sick; vomiting and purging came on, and continued through the night. On the following morning she appeared like one affected with malignant cholera. The skin was cold and dark-colored, the features contracted, and the breath cold; the pulse was small and wiry; there was restlessness, thirst, pain in the abdomen, and she lay in a sort of drowsy, half-conscious state. Whatever liquid was taken was immediately rejected, and the matters passed by stool consisted chiefly of a serous fluid with blood. She died in five days without rallying; the two other sisters recovered. On inspection, a large portion of the mucous membrane of the stomach was found abraded and softened in the course of the greater curvature. A similar case, in which three seeds destroyed the life of a man in forty-six hours, is reported in the *Med. Times and Gaz.*, May 25, 1861, p. 555. There was a general redness of the stomach, and the abraded portion presented the appearance of a granulating surface of a pale rose-color; it was covered by a considerable quantity of slimy mucus. The small intestines were inflamed, and the inner surface of them was abraded. The effects produced on the sisters who recovered, bear out the statement of Sir R. Christison, that two or three of the seeds will act as a violent cathartic. Other cases, including one which proved fatal, are recorded by M. Chevallier in the *Ann. d'Hyg.*, 1871, vol. 1, p. 400. A woman swallowed a quantity of the seeds bruised, in place of castor oil. She was soon seized with violent vomiting and bloody stools, which continued till her death on the fifth day after taking the seeds. The mucous membrane of the stomach and bowels was of a dark color, much ecchymosed, and it presented patches of small extravasations of blood.

An officer took as a purgative seventeen seeds. In three hours there was violent purging, followed by vomiting and severe cramps, the patient passing into a condition resembling the collapse of Asiatic cholera. The vomiting was not stopped until after twenty-one hours, and recovery then took place. There was suppression of urine for forty-eight hours (Wigger and Husemann's *Jahresbericht*, 1872, p. 538). The cake left after the expression of the oil, is poisonous to rats as well as human beings (Chevallier). When the seeds are swallowed whole, they may fail to produce the severe symptoms above described.

Mr. Little reports two cases of children, aged respectively six and three years, in which recovery took place, although the seeds had been masticated. The children when brought to the hospital were suffering from extreme collapse, consequent on vomiting and purging, the body pale and perspiring, pulse 130. The night before admission the children had eaten some castor-oil seeds. They suffered severely through the night. The stools were frequent and watery. The substance thrown from the stomach was pulpy; there was pain in the abdomen, great thirst, and the tongue was furred and dry. There were no cerebral symptoms. Under treatment, they both recovered in two days. (*Med. Times and Gaz.*, 1870, vol. 1, p. 581.)

FIG. 41.



Castor seeds.

Four other cases of recovery, in which, however, severe symptoms were induced, are reported by Dr. Pécholier, of Montpellier. In one of these three seeds only were taken. The symptoms were severe pain in the abdomen, coming on in an hour or two, copious and painful vomiting; burning heat; great thirst; convulsions, followed by collapse. M. Pécholier believes that there is no poison present in the seed, but that it is generated in the body by a reaction similar to that by which prussic acid and essential oil are produced from the pulp of the bitter almond. (*L'Empoisonnement par les Semences du Ricin*, 1869, p. 30.)

Analysis.—Castor *seeds* could only be identified in the contents of the stomach provided a portion of the outer coat could be obtained. These seeds are remarkable for their peculiarly variegated surface. Externally they are of a pale gray, but marbled with yellowish-brown spots and stripes.

COLCHICUM (MEADOW-SAFFRON).

Meadow-saffron (COLCHICUM AUTUMNALE) contains a poisonous alkaloid—*colchicina*—the effects of which on animals are similar to those of veratria, the alkaloid existing in white hellebore. The most noxious parts of the plant are the bulbs (or roots) and seeds, but the leaves and flowers have also an irritant action. Three deaths from colchicum are reported to have occurred in five years, 1863–7.

Symptoms and Appearances.—The effects of colchicum are those of a vegetable irritant: it causes a burning pain in the gullet and stomach, with violent vomiting, and occasionally violent bilious purging, followed by death. The general nature of the symptoms may be gathered from five fatal cases, which occurred at the Toulon Hospital under the treatment of M. Roux. (*L'Union Médicale*, Mars 27, 1855; and *Lancet*, May 5, 1855, p. 474.) It appears that *two ounces* of colchicum *wine* were given to each patient by mistake in place of bark wine. None of the men experienced any ill effects until about *two hours* after they had taken the poison. Two of them, who had felt an unusual burning at the stomach, with colicky pains, then began to vomit; and when examined, they were pale, cold, with a small pulse, and suffering from severe abdominal pain, nausea, constant vomiting, and frequent and abundant purging. In about six hours after the colchicum wine had been taken, there was a burning sensation in the throat and along the gullet, ardent thirst, and frequent yellowish serous evacuations, without mucus or blood. The mental faculties were unimpaired, and no alteration had taken place in the motor or sensory power. At five o'clock in the afternoon, nine hours after the colchicum had been taken, the symptoms were unchanged, except in one patient; in this case vomiting and purging had ceased, and the skin had become warm and moist. Three of the patients died after *nineteen hours'* suffering, and two after *twenty-six hours.* The two last were, shortly before death, in a deplorable state, presenting lividity of the lips and nails, icy skin, and heaviness about the head; they complained, besides, of a vesical and rectal tenesmus, great thirst, burning of the throat, and severe pains in the loins and limbs. The *appearances* were similar in the five patients, and were as follows: No ulceration or traces of inflammation in the throat and gullet; stomach and intestines distended with a little gas, but containing a great deal of opaque fluid; mucous membrane much softened and red, but presenting no ulcerations; liver considerably congested, and spleen gorged with blood. No other alteration existed, except a strongly marked redness of the brain and spinal marrow. The aspect of the muscular tissue was rosy in all parts, and three days after death decomposition had not begun. No colchicina was found in the vomited matters, but by comparative analyses, it was proved that the men had been poisoned by colchicum.

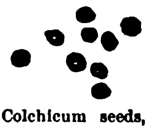
In November, 1839, a gentleman swallowed by mistake one ounce and a half of *wine* of colchicum. He was immediately seized with severe pain in the abdomen; other symptoms of irritation came on, and he died in seven hours. No examination of the body was made! In another instance, in which an ounce of the wine was taken, death occurred in thirty-nine hours. (Schneider's *Annalen*, vol. 1, p. 232.) In one case in which this dose was taken, the person recovered after suffering from cramps in the limbs, and twitching of the tendons. (*L'Union Méd.*, Aug. 24, 1848.) A woman, æt. 56, suffering from rheumatism, for whom wine of colchicum had been prescribed, took by mistake an ounce of the wine of the seeds, in divided doses, in twelve hours. She suffered from nausea, violent and profuse vomiting, slight purging, with heat and burning pain in the throat, great thirst, cold clammy skin, feeble pulse, pain in the stomach, and pain in the forehead. Inflammation of the stomach supervened, and the retching, vomiting, thirst, and pain continued for three days. She then recovered. (*Amer. Jour. Med. Sci.*, Jan. 1857; and *Brit. For. Rev.*, 1857, vol. 19, p. 409.) In a well-marked case of poisoning by the *wine* of colchicum, reported by Mr. Fereday, two ounces were taken. The symptoms did not come on for an hour and a half; there was then copious vomiting of a yellow fluid, severe pain with great tenderness in the abdomen, tenesmus and thirst. The patient died in forty-eight hours, without being convulsed or manifesting any sign of cerebral disturbance. The chief morbid appearance was a patch of redness in the mucous membrane of the stomach, near the cardiac orifice; the intestines were slightly inflamed. The head was not examined. (*Medical Gazette*, vol. 10, p. 161. See also Casper, *Ger. Med.*, p. 450.) A case of poisoning by the medicinal administration of colchicum has been communicated to me by Mr. Mann, of Bartholomew Close. Three and a half drachms of the wine of colchicum were taken in divided doses, and caused death on the fourth day. There was no inflammation of the mucous membrane, but simply extravasation of blood into the mucous follicles. The mucous membrane has been found softened in two cases of poisoning by the tincture. In two other cases, in which an ounce and a half of the *tincture* was taken, and death ensued in forty-eight hours, no morbid appearances were observed. (Casper, *Ger. Med.*, 1857, vol. 1, p. 451.) For a case of alleged poisoning by wine of colchicum, see Casper's *Vierteljahrsschrift*, 1860, vol. 1, p. 1.

Dr. Ollivier has published the details of two cases, in each of which about five ounces of the *tincture* of colchicum root were swallowed, and proved fatal. In one case there was continued and violent vomiting, but no purging. Pulse thready and slow; intense thirst; severe cramps in the soles of the feet; intellect unaffected; no convulsions or tetanic spasms. The patient died in twenty-two hours. The body was not inspected until putrefaction had advanced to a degree to destroy all the appearances. An unsuccessful attempt was made to extract colchicina from the contents of the stomach. In the other case, symptoms speedily appeared, indicated by violent pain in the abdomen; frequent vomiting, but no purging; difficult breathing; coldness of the skin; no tetanic spasms, but cramps in the soles of the feet; pulse small; intellectual faculties preserved. Death took place in twenty-eight hours. The vessels of the pia mater were much injected, but there was no redness of the mucous membrane of the stomach. (*Annales d'Hyg.*, 1836, vol. 2, p. 394.)

The mucous membrane has been found softened in two cases of poisoning by the tincture. In two other cases, in which an ounce and a half of the *tincture* was taken, and death ensued in forty-eight hours, no morbid appearances were found. (Casper, *Ger. Med.*, 1857, p. 451.)

Seeds.—A case is quoted in the *Pharmaceutical Times* (Jan. 23, 1847, p. 354), in which a man, æt. 75, swallowed a large quantity of colchicum *seeds*. He soon experienced a burning sensation in the throat, with nausea, vomiting, violent colicky pains, and frequent purging. These were succeeded by difficulty of breathing, and discharge of bloody urine. After death, patches of inflammation and mortification were found in the stomach and small intestine (duodenum). The latter contained some colchicum seeds.

FIG. 42.



Colchicum seeds, natural size.

The seeds of colchicum are of a brown color, varying from pale to dark. They are without smell, but have a bitter acrid taste. In size and color they somewhat resemble grains of paradise, and have been mistaken for them. Their shape and size are represented in the annexed engraving. (Fig. 42.) A man, æt. 52, took a *decoction* made with a tablespoonful of colchicum seeds to a pint and a half of water. He was seized with vomiting and purging, continuing incessantly until death, which took place in about thirty-six hours. The only appearance of note was that the stomach had a violet or purple hue. Two cases of death from the fresh seeds are reported in the *Journal de Chimie Méd.*, 1853, p. 421. The reader will find a good summary of the action of this plant in a paper by Dr. D. Maclagan. (*Ed. Month. Jour.*, December, 1851.) The roots, seeds, leaves, and flowers are poisonous, and cattle sometimes fall victims to its noxious effects. The poisoning of cattle by it is said to be frequent in Ireland. In October, 1874, twenty-three head of cattle were destroyed by reason of their being turned to graze in pastures where the colchicum was in flower. (*Pharm. Jour.*, Oct. 1874, p. 328.) In the spring, these animals suffer from the leaves.

Colchicum has acquired an evil notoriety as a poison, chiefly owing to the evidence given at a trial which took place at the Central Criminal Court, in September, 1862 (*Reg.* v. *Catherine Wilson*). This woman, after having been tried and acquitted for an attempt to poison, with oil of vitriol, the wife of a man with whom she cohabited, was in the following September convicted of the murder of a Mrs. Soames, who died suddenly while nursed by the prisoner six years previously. The body was exhumed, but no poison was found in the remains; yet the medical and other circumstances, as well as the conduct and correspondence of the prisoner, proved to the satisfaction of the court that deceased had been destroyed by vegetable poison, most probably colchicum, with the noxious properties of which she was proved to have been well acquainted. From the facts which transpired in reference to this trial, it appeared that the deceased was one of four persons who had at different dates fallen victims to the acts of this woman. 1st, Peter Mawer, a master mariner, of Boston, died in October, 1854; the body was exhumed in 1862, but no poison was detected. 2d, Mrs. Jackson, of Boston, who died in December, 1859; the body was exhumed in January, 1860, and no poison detected. 3d, Mrs. Atkinson, of Kirkby Lonsdale, who died in October, 1860; the body was exhumed in May, 1862, and no poison detected. 4th, the case of Mrs. Soames, above mentioned. All these persons died suddenly while in a state of health, under similar symptoms, and without any apparent natural cause to account for death. The symptoms as a whole were not reconcilable with any known disease; and they only appeared after the prisoner was proved to have administered, under some pretence or other, food or medicine, the bottle which she employed for this purpose being kept locked up, or in her own possession. The motive for the murder, in each case, was the acquisition of money or property of which the prisoner came into possession—in Peter Mawer's case by a will made shortly before his death, and in Mrs. Atkinson's case by an act of robbery after her death. Two other attempts at murder, which failed, led to the inference that colchicum seeds were employed by this woman, either in wine or brandy. In these four persons, the symptoms were as nearly as possible of the same character—burning pain in the throat and stomach, intense thirst, violent vomiting and purging, coldness and clamminess of the skin, excessive depression and great weakness. The pulse was small and weak, and death appeared to take place from complete exhaustion, without convulsions or loss of consciousness. Of these persons, one died on the second, one on the fifth, one on the eighth, and one on the fourteenth day from the occurrence of these symptoms. In most of the cases, the poison was probably given in divided doses; in the last case, the symptoms always appeared every evening after the deceased had taken the tea prepared by the prisoner.

Colchicum in the form of tincture or wine possesses a warm aromatic taste and a peculiar odor. The medicinal dose of the tincture and wine of colchicum is from ten to thirty minims, and of the powder from two to eight grains. According to Dr. Aldridge, the tincture, given frequently in medicinal doses, produces salivation. (*Dub. Hosp. Gaz.*, Oct. 1845, p. 52.)

Colchicina—Analysis.—Colchicum owes its noxious properties to the alkaloid *colchicina*, which exists in combination with an acid both in the seeds and root. It may be procured in fine white crystals, which have a bitter acrid taste. It is soluble in water, has a feeble alkaline reaction, and forms crystallizable salts with acids. In most of its reactions it resembles the other alkaloids. Its solutions have a bitter taste, give a white precipitate with tannic acid—a yellow with chloride of platinum, and a puce-brown with solution of iodine. The process for detecting this substance consists in neutralizing with potash a portion of the aqueous acid solution (obtained by evaporating the alcoholic tincture, or an alcoholic extract of the contents of the stomach), and then adding twice its volume of ether. The mixture should be well shaken. On pouring off the ether, and letting it evaporate spontaneously, an imperfectly crystalline or uncrystalline residue of a pale fawn color remains. When this is treated with one or two drops of concentrated nitric acid, it acquires a rich purple or reddish-violet color. This is the special test for colchicina. (*Pharm. Jour.*, 1857, p. 529.) Iodic acid produces no change in it. Sulphuric acid produces with it a dingy maroon color, and sulphomolybdic acid a reddish-black. A solution of colchicina is precipitated by the chloriodide of potassium and mercury, and the ioduretted iodide of potassium. It is a powerful poison. One-tenth of a grain killed a cat; vomiting, purging, and salivation were among the symptoms; these were followed by convulsions. The stomach and intestines were highly inflamed, and blood was effused throughout their course. (Pereira, *Mat. Med.*, vol. 2, pt. 1, p. 157.) In two cases less than half a grain proved fatal to adults. (Casper, *Ger. Med.*, 1857, vol. 1, p. 402.)

BLACK, WHITE, AND GREEN HELLEBORE.

There are several species of hellebore, but the two plants which are most commonly used as poisons under this name, are the black and white hellebore.

Black Hellebore.—This is the HELLEBORUS NIGER of the modern, and the MELAMPODIUM of the old pharmacopœias; it is also known under the name of *Christmas Rose*, from its flowering in January. In Lancashire it is called *Brank-ursine.* Another species, HELLEBORUS FŒTIDUS, sparingly grows in shady places and on a chalky soil, flowering in March and April; it is known under the names of *Bear's-foot*, *Setter-wort*, or *Helleboraster.*

Symptoms and Appearances.—According to Wibmer, the roots of the black hellebore possess great activity; but the leaves are also highly poisonous when used in the form of infusion. By long boiling the poisonous properties of the plant are diminished, probably owing to the loss of the volatile principle, which is an acrid oil. The roots and leaves have a local irritant action, producing violent vomiting and purging in small doses, with severe pain in the abdomen, followed by cold sweats, convulsions, insensibility, and death. The powdered root, in a dose of a few grains, acts like a drastic purgative. In a case reported by Morgagni, half a drachm of the aqueous extract killed a man, aged fifty, in eight hours. The symptoms were severe pain in the abdomen and violent vomiting. After death the whole of the alimentary canal was found inflamed, but especially the large intestines. (Wibmer, *op. cit.*, HELLEBORUS.) A case is quoted by the same writer, in which a tablespoonful of the finely powdered root (taken by mistake for rhubarb) caused severe symptoms of irritant poisoning, which did not disappear for four hours. The man recovered on the fourth day. The experiments performed by Orfila on animals show that this poison acts like a *local* irritant when applied to a wound on the skin. (*Op. cit.*, vol. 2, p. 369.)

In December, 1862, Dr. Edwards met with a case in which a gentleman had swallowed experimentally one drachm of tincture of green hellebore (*veratrium viride*), equal to twelve grains of the powder. He was found soon afterwards in a collapsed state, features sunk, skin cold, and covered with a profuse clammy sweat, pulse scarcely perceptible. He complained of intense pain in the region of the stomach. There was no purging. These symptoms were relieved by treatment, and the next morning the patient had recovered. (*Med. Times and Gazette*, 1863, vol. 1, p. 5.)

In the following case, which occurred to Dr. Massey, a woman recovered from a large dose of the infusion. About one ounce and a half of the root of the black hellebore was put in a covered jar in an oven with twelve ounces of water; it was allowed to remain a whole night by a slow fire. On the following morning the woman took a teacupful of the infusion. It produced pain and pricking in the tongue, fauces, and throat; to use her own expression, "as if a hundred pins were pricking her." There was a painful sense of constriction of the throat, with great difficulty of swallowing; pain at the stomach, and violent sickness. The tongue began to swell, as well as the parts about the throat, and much viscid mucus was voided from the mouth. The eyes were sunk; there was excessive prostration of strength, discoloration about the eyelids, with great collapse of the vital powers, such as is seen in the collapsed stage of Asiatic cholera; the extremities were cold, and the general surface of the body was bedewed with a cold clammy sweat. The pulse varied from thirty to fifty beats in a minute, was very small, and at times scarcely perceptible. An emetic of sulphate of zinc was given with a large quantity of lukewarm water; and this was followed by three grains of camphor dissolved in spirits of wine, and mixed with yolk of egg. The skin was kept warm by hot applications. Coffee was repeatedly given; and in the course of three or four hours the patient rallied considerably. The pulse rose to sixty-eight and seventy. She complained of headache; the bowels were relieved with castor oil; and beyond saline effervescing draughts nothing further was required. She soon recovered. (*Lancet*, July 26, 1856, p. 100.)

Hellebore is a favorite remedy for worms with quacks and rural doctresses. It is not, therefore, surprising that it should be occasionally administered in an overdose and cause death. Mr. Todd, Coroner for Hants, forwarded to me the report of an inquiry which took place before him in November, 1845, in which a child under two years of age was poisoned with an *infusion* of hellebore, administered to it by its grandmother, for the purpose of destroying worms. The leaves of the plant (Bear's-foot) were bruised, and boiling water poured over them. Two dessertspoonfuls were given to the child, who had been suffering from ague, but from which he had recently recovered. Within ten minutes after taking the mixture he was very sick, and was violently purged. The matter vomited was of a green color, and slimy; the sickness and purging continued until the evening, when he died, *i. e.*, about thirteen hours after having taken the mixture. There were convulsions before death. On inspection, the whole body appeared blanched; the eyes were sunk, and the pupils dilated. There was diffused inflammation of the mucous membrane of the stomach, and a well-marked patch of inflammatory redness, about the size of a five-shilling piece, near its centre. The small intestines, which contained a brownish-yellow fluid, were much inflamed. The cæcum contained about thirty worms. The head and chest were not examined. Death was very properly attributed by the medical witness to the action of hellebore. The woman who prepared the infusion stated that she had frequently given it in large quantities to children, and there were no injurious effects. It is nevertheless to be regarded as an active poison; and if persons are not always killed by such worm-medicines, it must be considered a very fortunate circumstance. This acrid vegetable never can be given by an ignorant person without great risk.

Analysis.—This is confined to the botanical characters of the leaves and roots. Black hellebore has a large flower with five round spreading petals, which are at first white, and afterwards become reddish-colored, and finally greenish. The flower of fetid hellebore, or bear's-foot, has five oval concave petals, of a green color, tinged with purple at the margin.

White Hellebore—Veratrum Album.—The action of this plant is analogous to that of black hellebore; it is, however, more irritant. The powdered root produces a strong local effect, and causes violent sneezing. When taken internally, it gives rise to severe pain in the abdomen, vomiting and purging, followed by giddiness, dilatation of the pupils, convulsions, insensibility, and death. It produces a sense of great heat and constriction in the throat. In three cases mentioned by Dr. Pereira, in which the infusion had been swallowed, there was no purging. (*Op. cit.*, vol. 2, part 1, p. 170.) A man took by mistake half an ounce of white hellebore in powder. Four hours after taking the poison he was seen by a medical man. The chief symptoms were, a burning sensation in the mouth and throat, pain in the stomach, purging, nausea, but no vomiting until after an emetic had been given, There were no symptoms affecting the brain. Under treatment the man soon recovered. (Mr. Giles, in *Lancet*, 1857, vol. 2, p. 9.)

There can be no doubt that white hellebore is an irritant poison. The numerous observations collected by Wibmer prove that it acts powerfully on the system. In one instance, twenty grains of the powder caused convulsions and death in three hours, and in another, a man, after eating the root, died in six hours. Death was preceded by vomiting of bloody mucus, and by cold sweats. (*Op. cit.*, VERATRUM.) The smallest quantity required to destroy life is unknown. Sir R. Christison quotes a case from Bernt, in which a man took but a small quantity of the powder, and died in the course of the day. After death, the same marks of irritation were found in the alimentary canal as those which have been described in treating of black hellebore.

Analysis.—Powdered *white hellebore* root has a reddish-brown color, resembling jalap. Nitric acid gives to it a red, rapidly passing to a dark-brown, color. Sulphuric acid produces with it a dark-brown tint, almost black. Iodine water, a bluish-gray tint, slowly brought out. The proto and persalts of iron have no effect upon it.

Veratria.—White hellebore owes its noxious properties to the alkaloid *veratria*, which is itself a powerful poison. The late Mr. Callaway communicated to me the following case. A physician prescribed medicinally for a lady one grain of veratria divided into fifty pills, and three were directed to be taken for a dose. Not long after this dose had been swallowed, the patient was found insensible, the surface cold, the pulse failing, and there was every symptom of approaching dissolution. She remained some hours in a doubtful condition, but ultimately recovered. Supposing the medicine to have been well mixed, and the pills equally divided, not more than one-sixteenth of a grain of veratria was here taken! The common veratria of the shops is sometimes given medicinally, in doses of one-sixth of a grain. Poisoning by veratria is a rare occurrence. I have not met with an instance in which this alkaloid has been administered with criminal intention. With the exception of the case above mentioned, there is no experience of its operation as a poison on man. Judging from its effects on animals, it would cause vomiting and convulsions, with insensibility.

Analysis.—In the state in which it is usually seen *veratria* is a brownish-white uncrystalline powder, scarcely soluble in boiling water, but dissolved by alcohol, ether, and benzole. Acids readily dissolve it, forming salts which on evaporation do not yield crystals. The powder has a hot, acrid taste, without bitterness, and if any portion enters the nostrils, it produces a copious flow of mucus and the most violent sneezing, lasting for some time. It enters into the composition of most cephalic snuffs. In its local action it is powerfully irritant. Strong nitric acid gives to the powder a light-red color, becoming ochreous after a time. Hydrochloric acid, strong and diluted, with the aid of heat, produces a dingy red tint. The best test for its presence is the diluted sulphuric acid, which dissolves it without change, and by a gentle heat produces a rich pink color, which is destroyed by a solution of chlorine, but not by chloride of tin. Strong sulphuric acid turns the powder yellow, but on heating the mixture the color deepens, and finally becomes of a deep maroon red. When the liquid is diluted, this color changes to a dingy yellow. Iodic acid dissolves it without change. Sulphomolybdic acid turns it of a pale reddish-brown.

Veratria differs from *colchicina* in its insolubility in water, as well as in the action of strong nitric and diluted sulphuric acid. Veratria may be brought into solution in organic liquids, by acetic acid and heat. The liquid is treated with potash, and two parts of benzole will yield the alkaloid if present, on decanting and evaporating the benzolic solution. The tests may then be applied to the residue. It has not yet been detected in the tissues upon any reliable authority.

A case occurred in September, 1865, in which death was attributed to the action of veratria criminally administered. The deceased, a woman, was advanced in pregnancy, and from the medical evidence she died from puerperal convulsions, and Bright's disease of the kidney, with effusion of blood on the brain. It was said that veratria was detected in her body and in the urine, but there were no symptoms of poisoning by veratria, and there was no evidence of administration by any one. The chemical analysis was not published, or it might have appeared that too great a reliance had been placed upon the tests employed. (*Med. Times and Gazette*, October 28, 1865, p. 472.) It was no doubt a case of death from natural causes mistaken for poisoning. It may here be observed that Selmi has lately found in the viscera a principle which is likely to give rise to fallacious results, resembling those assigned to veratria. It is precipitated by some of the precipitants of the alkaloids, and acquires a violet-red color with sulphuric acid when gently heated. (Wiggers, *Jahresb.*, 1874, p. 599.) This has no doubt misled very confident analysts into the belief that they had detected veratria in the viscera.

ERGOT OF RYE (SECALE CORNUTUM).

This substance is better known as an abortive drug than as a poison. When taken in one large dose, or in small doses long continued it operates as an irritant. It is a diseased growth in the grain or seed of rye caused by a parasitic fungus. In powder, infusion, or tincture it has been for some time used by medical men to excite the muscular action of the uterus and aid parturition. It is also used for a similar purpose on animals in veterinary practice.

Symptoms and Appearances.—In large doses ergot has been found to cause dryness and irritation of the throat, thirst, burning pain in the stomach, vomiting, colic, and sometimes purging. Cerebral symptoms, such as headache, giddiness, and stupor have also been met with. The appearances after death have been, in the few cases recorded, chiefly patches of inflammatory redness in the mucous membrane of the stomach and small intestines. In doses of from half a drachm to two drachms ergot in powder has caused nausea, vomiting, dryness of the throat, great thirst, aversion from food, pain in the abdomen, slight purging, pain in the head, stupor, and dilatation of the pupils. (Pereira, *Mat. Med.*, vol. 2, pt. 1, p. 111.) The medicinal dose of the powder in uterine diseases is from 5 to 15 grains. It is employed in a larger dose (from 20 to 60 grains at intervals of half an hour) to excite uterine action either for abortion or parturition. The dose of the tincture is from ten minims to one drachm (a teaspoonful), which is considered to be equivalent to 20 grains of the powder. The dose of the ethereal tincture, according to Pereira, when employed for the purpose of exciting uterine action, is one drachm every half hour for three or four doses. (*Mat. Med.*, vol. 2, pt. 1, p. 112.) Ergot must be regarded as a noxious substance, and by some authorities, it is ranked among narcotico-irritant poisons. It does not easily cause death in one large dose, but its fatal operation appears to be more strikingly developed by its long-continued use in small or medicinal doses.

A case occurred at Brighton in October, 1864, in which a question arose respecting the fatal effects of this drug on a woman who had taken it for a long period, obviously with a view to procure abortion. She died, however, without abortion having taken place; and the question at issue was, whether this drug had or had not caused her death. The dose taken was, I am informed, about a teaspoonful of the tincture of ergot three times a day for a period of eleven weeks. On inspection, patches of inflammation were found on the mucous membrane of the stomach after death. No other cause for death was apparent, and one medical witness assigned it to the irritant action of the ergot, as at the early stage of pregnancy which she had reached (the third month), this substance would not be likely to act on the uterus. Another medical gentleman, who gave evidence at the inquest, asserted that death could never be *primarily* caused by ergot of rye. The qualification introduced into this medical opinion is of small importance. The deceased woman is reported to have taken a large quantity of the tincture, and it is quite immaterial whether the drug killed her by a primary or secondary operation. M. Tardieu describes the case of a woman, æt.

24, who aborted in the fourth month of pregnancy, as a result of the administration of ergot in powder; she died from peritonitis in about twenty-four hours. The ergot was found in fragments in the lower third of the bowels. (*Ann. d'Hyg.*, 1855, vol. 1, p. 404.)

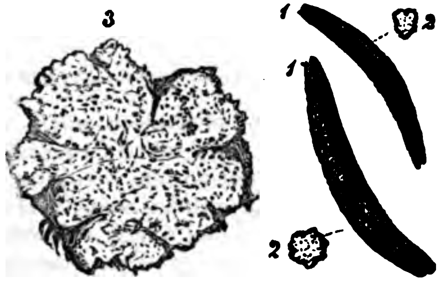
In respect to its operation, it may be observed that the effects produced by its administration are not such as readily to excite suspicion. It does not cause the decided symptoms of irritation observed in the action of savin. In medicinal doses, given at proper intervals, the only marked effect which it produces on a pregnant woman is a lowering of the pulse. Sometimes other symptoms of a severe character have presented themselves. (*Ann d'Hyg.*, 1856, vol. 1, p. 140.) If a person dies from the effects of this drug, the results are legally the same, whether its operation as a noxious substance is of a primary or secondary kind.

Small doses of the aqueous extract of ergot injected beneath the skin in animals, caused a loss of sensation and of the power of co-ordination; larger doses produced paralysis both of voluntary and reflex motions, and with paralysis, convulsions occur. The pupils are dilated. The pulse is at first quickened, but it afterwards decreases until death. The active principle is soluble in water, but not in alcohol. (*Med. Record*, Jan. 7, 1873.)

The *chronic* effects of this poison have been witnessed occasionally on the continent in an epidemic form, and they have in some instances been distinctly traced to the admixture of ergot with rye-bread. In one set of cases the nervous system was found to be especially affected, indicated by vertigo, loss of sensation, somnolency, rigidity of the muscles, tremulous gait, and convulsions. After death the chief appearances were congestion of the brain, liver, and lungs. In another set of cases the blood appeared to have undergone some remarkable change; hemorrhages ensued; black spots and boils appeared in various parts of the body, and gangrene took place in the extremities. After death the blood was found black and very fluid throughout the body. (Wibmer, *op. cit.*, art. Sphacelia Segetum, p. 158.) These serious effects are not witnessed in this country, because rye-bread is but little used as an article of food. The presence of ergot in bread may sometimes account for the symptoms of poisoning which have been observed. (*Dublin Med. Press*, July, 1847, p. 53.) Much information on this subject will be found in the following references: (*Ann d'Hyg.*, 1834, vol. 2, p. 179; 1835, vol. 2, p. 240; 1843, vol. 1, p. 347; Henke, *Zeitschrift der S. A.*, 1842, vol. 2, p. 185; 1844, vol. 1, p. 286, and vol. 2, p. 215.) It would appear that on the continent this condition, to which the name of *Ergotism* has been given, requires for its production a long-continued use of the diseased grain.

Analysis.—The active properties of ergot have been ascribed to the presence of an oil or oleo-resin, soluble in ether. According to Herrmann an ethereal solution, distilled, yields a fixed oil of a brownish-yellow color, of aromatic odor and an acrid taste; it exerts no particular action, either as a medicine or a poison. An alkaloid called *ecbolin* has been discovered associated with the oil, and separable from it by a complex process (see *Pharm. Journ.*, Sept. 1871, p. 242); to this alkaloid, according to Wenzell, the medicinal action of ergot is due; others regard ecbolin as inert. (*Med. Record*, Jan. 1873.) Ergot is soluble in water and alcohol, and the aqueous is more powerful than the alcoholic or ethereal extract. The active principle is dissolved by water, but only to a slight extent by alcohol. In addition to ecbolin Husemann describes another alkaloid, *ergotin*, and gives very elaborate processes for their separation from ergot and from each other. (*Die Pflanzenstoffe*, 1871, p. 521.) They are brown amorphous substances, faintly bitter to the taste, and having an alkaline reaction. They are soluble in water and alcohol, but insoluble in ether and chloroform. They combine with acids to form amorphous salts. A solution of ecbolin gives a white precipitate with cyanide of potassium. Ergotin gives no precipitate with the cyanide. Sulphuric acid dissolves ecbolin, giving to it at the same time a dark maroon color.

Fig. 43.



The ergot of rye.

The form and characters of the ergot in mass are well known to professional men. It consists of grains varying in length from half an inch to an inch and a quarter, and the breadth of about the eighth of an inch. The grain is cylindrical, blunt at the ends, and curved like the spur of a cock. The outer coat is of a dark purple color, almost black, irregularly fluted on the surface, which is often irregularly cracked and fissured. In the annexed illustration (Fig. 43) 1 1 represent the ergot of rye as it is usually seen. The smaller of the two grains represents the average size; 2 2 are sections of the grains, and 3 represents a transverse section, magnified thirty diameters. The spongy character of the substance of the ergot is here more distinctly seen.

The powder of ergot has a faint fishy smell; this is especially observed when it is rubbed with a solution of potash. This alkali dissolves it in part, and the solution acquires a dingy-red or claret color, owing to the solvent action of the alkali on the coloring matter of the outer coat. In the form of tincture, alcoholic or ethereal, the peculiar fishy odor of the extract when treated with potash is well marked. This is considered to be owing to the production of propylamin. It may, however, be concealed by other odors. Sometimes small particles of ergot, presenting a pink-red color in the dark external coat, may be detected in the sediment by the microscope. When ergot has been taken in powder, fragments of it may be found scattered over the lining membrane of the stomach or bowels; these may be identified by the characters above described. The ethereal tincture of ergot, evaporated to an extract, yields a yellowish-colored oil, which, if any of the coloring matter of ergot is present, acquires a reddish color when heated with a solution of potash. On the chemical and microscopical properties of ergot, see a paper by Dr. Lex. (Horn's *Vierteljahrs.*, 1866, vol. 1, p. 231.) For the purpose of applying tests, it is advisable to obtain an alcoholic extract and dissolve this in water. Alkalies give to the solution a crimson tint, and the sulphates of copper and zinc throw down purplish-colored precipitates. (*Pharm. Jour.*, 1858, p. 511.)

It is not probable that a sufficient quantity of this substance will be found in the body of a person to whom it is alleged to have been given, to allow of the separation of *ecbolin* or *ergotin*. The medical jurist must rely upon the physical properties of the fungus if he can obtain any of it. A spectral examination of the red alkaline solution of coloring matter presents nothing characteristic. The dry powder, heated in a reduction-tube, yields nitrogen as ammonia, and sulphur as sulphuretted hydrogen, discoverable by red litmus and lead-paper. Old samples smell strongly of ammonia, and contain often living acari.

ALOES—COLOCYNTH—GAMBOGE—JALAP—SCAMMONY.

These different substances, which are used in small doses as medicines, are liable, when taken frequently or in large quantities, to excite, in the stomach and bowels, vomiting, purging, and other symptoms of irritation. Colocynth has occasioned death in several instances; in one case a teaspoonful and a half of colocynth powder destroyed life; and one drachm of gamboge, a medicine much used by quacks, has proved fatal to man. (Traill's *Outlines*, p. 150.) Aloes and colocynth mixed, are said to be the basis of a certain quack medicine sold under the name of *Morison's Pills*. These have proved fatal in several instances from the exhaustion produced by excessive purging, owing to the large quantity of the pills taken in frequently repeated doses. Our knowledge of the symptoms and appearances produced by these irritants is, indeed, chiefly derived from the cases which have proved fatal under this pernicious treatment. In the seventeenth volume of the *Medical Gazette* will be found four cases of this description. The most prominent symptom is excessive purging, with the discharge of large quantities of mucus; the individual becomes exhausted, and slowly sinks. In some instances, the symptoms are those of inflammation and ulceration of the bowels. In 1836, a man was convicted of having caused the death of a person by the administration of these pills; in this instance, the death of the deceased was clearly due to the medicine, and on inspection, the stomach was found inflamed and ulcerated; the mucous membrane of the small intestines was inflamed and softened, and there was the appearance of effused lymph upon it. An ingenious attempt was made in the defence to draw from the medical witness a statement that the good effects of some medicines invariably increased in proportion to the quantity taken. This theory was, however, very properly rejected. The same remarks apply to *Holloway's Pills*, although these are of a more innocent description. The principal ingredient in them is said to be aloes. In all cases it must be remembered that these drastic purgatives may cause dangerous symptoms, or even death, when administered to infants, or to persons debilitated by age or disease; and it is not necessary that the dose should be very large, in order that fatal effects should follow. The question here will be whether the medicine caused death, or whether it simply accelerated it; although, in a legal point of view, that which accelerates, causes.

In March, 1875, a case in which a large dose of *colocynth* destroyed life, occurred at Plaistow. I am indebted to Mr. D. Price for the following details: A woman, æt. 38, took 120 grains of colocynth in powder. She believed she was pregnant, and took the powder in order to produce miscarriage. It brought on violent retching and purging, which continued more or less for fifty hours, when she died from exhaustion. When seen the day after she had taken the colocynth, there was great prostration of strength, with cold extremities, and a very weak pulse.

Hierapicra (*Holy bitter*) is a popular aloetic compound, and one death is recorded to have been produced by it in 1837–8. There is reason to believe that it is occasionally used for the purpose of procuring criminal abortion. A man was tried and convicted of this crime at the Aylesbury Lent Assizes, 1857 (*Reg.* v. *White*), and the noxious properties of this compound then became a subject of inquiry. The dose, and the condition of the woman to whom it is administered, will of course affect the answer to this question. At the trial above mentioned, it was properly considered to be a noxious substance within the meaning of the statute. The fact that, under the name of *Aloes cum Canellâ*, it was formerly admitted into the British Pharmacopœias, cannot justify the mischievous uses to which it may be put. *Hierapicra* is a snuff-colored powder, of an intensely bitter taste. In consists of four parts, by weight, of aloes, and one part, by weight, of powdered Canella bark. The proper medicinal dose was formerly fixed at from five to fifteen grains. Its injurious effects on pregnant women are chiefly due to the aloes. This specially affects the rectum, and, by contiguity, under violent irritation or purging, may affect the uterus. From the taste and color which it imparts to liquids, it is not probable that it could be taken by a woman unknowingly.

Death has been caused by aloes taken in nitric acid; but in this case the mineral acid was most probably the destructive agent. A singular case occurred in Germany a few years since, wherein a medico-legal question was raised respecting the poisonous properties of *Aloes*. A woman, æt. 43, not laboring under any apparent disease, swallowed two drachms of powdered aloes in coffee. Violent purging supervened, and she died on the following morning, twelve hours after having taken the medicine. On inspection the stomach was found partially, and the small intestines extensively, inflamed. There were no other particular appearances to account for death, and this was referred to the effect of the aloes.

Guinea pepper, *Grains of Paradise*, is a substance which has been elsewhere described as innoxious (*ante*, p. 486). It is a warm stimulant. Although not injurious, brewers have been justly condemned to penalties for putting it into beer as an adulterant.

Other vegetable irritant substances might be enumerated among poisons, but I believe these are the principal which have given rise to, or are likely to give rise to, medico-legal inquiries.

ANIMAL IRRITANTS.

CHAPTER LIII.

ANIMAL IRRITANTS—CANTHARIDES OR SPANISH FLIES—SYMPTOMS AND EFFECTS—ANALYSIS—NOXIOUS ANIMAL FOOD—FISH—MUSSELS—CHEESE—SAUSAGES—DISEASED FLESH OF ANIMALS—TRICHINOSIS—POISONED GAME.

CANTHARIDES (SPANISH FLIES).

Symptoms and Effects.—Cantharides are not unfrequently administered, either in the state of powder or tincture, for the criminal purpose of procuring abortion, but they are not often a cause of death in this country. Out of 1620 fatal cases of poisoning in five years, there were only two which were ascribed to cantharides. When taken in *powder*, in the dose of one or two drachms, this substance gives rise to the following symptoms: a burning sensation in the throat, with great difficulty of swallowing, violent pain in the abdomen, with nausea, and vomiting of a bloody mucus; there is also great thirst and dryness of the throat, and in a few cases observed by Mr. Maxwell, salivation was a prominent symptom. As the case proceeds, a heavy dull pain is commonly experienced in the loins, and there is an incessant desire to void urine, but only a small quantity of blood or bloody urine is passed at each effort. M. Lavallée found that one effect of this poison, externally applied, was to give a strongly albuminous character to the urine. (*L'Union Médicale*, June 17th, 1847, p. 380.) The abdominal pain becomes of a violent griping kind. Purging supervenes, but this is a symptom which is not always observed; the matters discharged from the bowels are mixed with blood and mucus, and there is often tenesmus (straining). In these, as well as in the vomited liquids, shining green or copper-colored particles may be commonly seen on examination, whereby the nature of the poison, if it has been taken in powder, will be at once indicated. After a time, there is severe priapism, and the genital organs are swollen and inflamed both in the male and female. In one instance, observed by the late Dr. Pereira, abortion was induced, probably owing to excitement of the uterus, from the severe affection of the bladder; for there is no proof that this substance acts directly on the uterus to induce abortion. With respect to the aphrodisiac propensities said to be caused by cantharides, these can seldom be excited in either sex, except when the substance is administered in a dose which would seriously endanger life. When the case proves fatal, death is usually preceded by faintness, giddiness, and convulsions.

The *tincture* of cantharides produces similar symptoms. They are, however, more speedily induced, and the burning sensation and constriction of the throat and stomach are more strongly marked; this symptom is often so severe as to render it impossible for the person to swallow; and the act of swallowing gives rise to excruciating pain in the throat and abdomen. Cantharides have been in some cases wantonly used, and with great danger to life, with a view of exciting sexual feelings. The doses in which it has been given, have been such as to cause symptoms of irritant poisoning. In November, 1859, six female servants in a gentleman's family, as well as the master and mistress, were attacked with all the symptoms of poisoning by cantharides. It appeared that the coachman of the family had, shortly before the occurrence, purchased an ounce of cantharides; that he had put the powder into beer and coffee, and had thus poisoned the whole household. He was tried, but acquitted of any indictable offence, on the ground that his intent was not to murder. It was this case which led to an immediate alteration in the law (p. 19, *ante*).

The following is a well-marked case of poisoning by the *tincture.* A boy, æt. 17, swallowed an ounce. When seen, an hour and a half afterwards, the respiration was hurried, there was profuse salivation, convulsive trembling, acute pain in the regions of the stomach and bladder, and such exquisite sensibility that the slightest pressure produced convulsions. These came in paroxysms, were accompanied by painful priapism, and followed by delirium. On the seventh day he was seized with pain in the head, trembling and universal spasms; coma followed. He then appeared to improve; but on the fourteenth day violent convulsions supervened, and these were followed by insensibility and death. (*Beck's Med. Jur.*, 5th ed. p. 842.) A woman swallowed a similar quantity of the *tincture.* Some time afterwards she suffered from severe pain in the abdomen, increased by pressure; it became swollen and tympanitic. She passed during the night a pint and a half of urine unmixed with blood. In two days, the pulse was feeble, and scarcely perceptible; there was delirium, with severe pain in the region of the kidneys and bladder; the urine was continually drawn off by a catheter. It was more than a fortnight before she was convalescent. (*Med. Gaz.*, vol. 29, p. 63.) A man swallowed sixty grains of the *powder* of cantharides by mistake for jalap. Some hours afterwards he was found laboring under incessant vomiting, intense thirst, with burning pain in the mouth, throat, and stomach, countenance anxious, tongue swollen and thickly coated, pulse 130, weak and tremulous; the matter vomited had a greenish color, and a peculiarly offensive odor. There were frequent and urgent calls to micturition, always preceded by severe pain at the point of the penis; and the passage of the urine was attended with severe scalding. The urine was turbid, and slightly tinged with blood. There was a dull heavy pain in the lumbar region, increased by pressure, and occasional priapism. Vomiting was promoted, and a large quantity of a thick solution of gum-arabic was administered at intervals. The patient rapidly recovered; his recovery was probably due to the greater part of the poison having been ejected by the early occurrence of vomiting. (*Med. Gaz.*, vol. 39, p. 385.)

A woman took a piece of blistering plaster, containing cantharides, about the size of a walnut, by mistake. In about an hour, vomiting and strangury supervened; these symptoms were followed by inflammation of the kidneys. She speedily recovered. In another instance, in which half an ounce of the plaster, containing two drachms of the powder, was taken by a lunatic, æt. 45, death took place in twenty-four hours, although remedies were immediately applied. In about two hours, the whole of the mucous membrane of the mouth was reddened, and covered with small blisters. In seven hours there was great coldness of the surface, with imperceptible pulse. The urine passed was mixed with blood. (*Ed. Med. and Sur. Jour.*, Oct. 1844, p. 563.) A singular case, in which an attempt was made to poison a man with blistering plaster, was the subject of a trial in France. This person perceived, after taking some soup, a strong and bitter taste, for which he could not account. He also suffered from violent pain in the stomach and abdomen, especially in the region of the bladder; and he could only, under scalding pain, void a small quantity of urine, tinged with blood. He recovered from these symptoms; but three months subsequently, and two hours after taking some soup which had the same bitter taste, they returned in an aggravated form. They were relieved by doses of olive oil and milk to excite vomiting. A few days afterwards, he found in this soup a dark-colored substance, which, on examination by a medical man, turned out to be the powder of cantharides. His brother-in-law, who was proven to have recently purchased blistering plaster, was tried on a charge of attempting to poison him. One-half of the plaster sold to the prisoner was found, and it was proved to contain about thirty-one grains of powdered cantharides. The medical witnesses agreed that the symptoms under which the prosecutor had labored were those which commonly result from this poison; but one of them contended that the dose administered was not sufficient to cause death! (The exact quantity taken is not stated.) The accused was nevertheless capitally convicted. (*Journal de Chimie*, 1846, p. 606.)

Chronic Poisoning.—It is not often that we have occasion to observe poisoning with cantharides in a chronic form; but a remarkable set of cases has been reported by Mr. Frestel, which show that, contrary to common belief, the substance does not invariably excite those aphrodisiac propensities which have been generally ascribed to it. It appears that six young men (students) had, during a period of six months, unknowingly taken with their food powdered cantharides by mistake for pepper. The quantity taken was at no time large, but very variable. The only marked general symptom was great restlessness. There was no affection of the nervous system, nor any disorder of the bowels. The appetite was unaffected. No pain was experienced in the renal or lumbar regions. About three hours after the meal, there was a slight pruritus of the glans, with a desire to micturate, and there was also *ardor urinæ*. The desire for micturition continued for from two to four hours, and then gradually ceased, leaving some irritation about the urethra. There was neither priapism nor any erotic feeling. The absence of symptoms is the more remarkable, as the substance must have been taken in very different doses at different times. Without knowing the cause of the disorder from which they had suffered, they employed for their relief, warm baths and an abundance of warm demulcent drinks. The plan of treatment was found to be effectual. (*Journal de Chimie Médicale*, Janvier, 1847, p. 17.)

Effects of External Application.—Cantharides will operate as a poison when applied externally to a wound, or ulcerated surface, or even when applied to a large surface of healthy skin. In January, 1841, a girl, æt. 16, was killed at Windsor under the following circumstances. She was affected with the itch: Sulphur ointment was prescribed for her, but, by mistake, blistering ointment was used. This was rubbed all over the body of the girl; she was soon seized with violent burning pain; the ointment was immediately washed off, but the cuticle came off with it. The girl died in five days, having suffered from all the usual symptoms of poisoning with cantharides.

Appearances after Death.—In one well-marked case of poisoning by cantharides, the whole of the alimentary canal, from the mouth downwards, was in a state of inflammation, as well as the ureters, kidneys, and internal organs of generation. The mouth and tongue seemed to be deprived of their mucous membrane. In another instance in which an ounce of the tincture was swallowed, and death did not occur for fourteen days, the mucous membrane of the stomach was not inflamed, but it was pulpy and easily detached. The kidneys were, however, inflamed. The brain has been found congested, and ulceration of the bladder is said to have been met with. There are very few fatal cases reported, in which the appearances have been accurately noted; indeed, the greater number of those who have taken this poison have recovered. In one fatal case, on an inspection of the body, the vessels of the brain were found gorged with blood, and a quantity of serum was effused in the ventricles and between the convolutions. The heart and lungs were healthy. The internal surface of the stomach was studded with red points interspersed with ecchymosis, in the centre of each of which was seen an adhering particle of the powder of cantharides. The intestines were healthy, but the kidneys were red and gorged with blood. The left ureter internally was of a very red color. The bladder was thickened, and the mucous coat injected with blood. (*Ed. M. and S. J.*, Oct. 1844, p. 563.) In a case which occurred to Mr. Saunders, death took place in about twenty-four hours. The deceased must have taken the greater part of half an ounce of cantharides in powder. The symptoms were such as have been above described. On inspection, the vessels of the brain were filled with dark-colored blood, and the ventricles were distended with serum. Both lungs were highly engorged with dark-colored blood. The gullet was partially inflamed, and there were patches of inflammation on the mucous coat of the stomach which had become detached in several places. The same inflammatory appearance existed in the small intestines, in the folds of which the powder of cantharides was abundantly seen.

The vessels were distended and the liver was engorged with dark blood. The gall-bladder was much distended with bile, and none of this secretion appeared to have passed into the bowels. The spleen and kidneys were highly congested; the ureters were inflamed; the bladder contracted and empty, and its internal surface pale. The glittering of the particles of cantharides in the viscera during the inspection by candle-light was very remarkable. (*Med. Times*, Feb. 2, 1849, p. 287.)

Cantharides are sometimes described as a corrosive poison; but the substance appears to have no local action of a chemical nature. It is a pure *irritant*, and the local effects observed are entirely due to irritation and inflammation. Serious accidents have frequently occurred from the powder of cantharides having been mistaken for jalap, cubebs, and other medicinal substances. A man lost his life on one occasion, by having cantharides powder supplied to him for cubebs in a druggist's shop.

Fatal Dose.—The quantity of this poison required to produce serious symptoms or to destroy life has been a frequent subject of medico-legal inquiry. Dr. Thomson represents the medicinal dose of the powder to be from one to three grains. On a late criminal investigation a medical witness stated that one grain was the maximum dose, but this, according to Thomson, is an under-statement; the dose of the tincture is from ten minims gradually increased to one fluid drachm—of the powder from *one to two grains*. (Pereira, *Materia Medica*, part 2, vol. 2, p. 754.) Doses above this, whether of the powder or the tincture, are likely to prove injurious, and to give rise to symptoms of poisoning. On a trial which took place at Aberdeen, in 1825, it appeared that a drachm of the powder had been administered: severe symptoms followed, but the person recovered. Dr. Dyce, the medical witness, said he had given ten grains of the powder as a medicinal dose. In three cases, observed by Mr. Maxwell, a drachm of the powder mixed with six ounces of rum was taken by each person; they were robust, healthy negroes; they suffered severely, but recovered in about ten days. In these cases, irritation of the urinary organs did not appear until after the men had been bled.

The *smallest quantity* of the powder which has been known to destroy life, was in the case of a young woman, quoted by Orfila; the quantity taken was estimated at *twenty-four grains* in two doses. She died in four days; but as abortion preceded death, this may have been concerned in accelerating that event. Her intellect was clear until the last. In one instance a man recovered after having taken twenty grains of the powder (*Ed. Med. and Surg. Journal*, October, 1844); and in another, after having taken *two drachms* (*Med. Gaz.*, vol. 42, p. 873). An *ounce* of the *tincture* has been known to destroy life. This dose was taken by a boy, æt. 17, and he died in fourteen days. This, I believe, is the smallest dose of the tincture which has proved fatal. Four drachms, and even six drachms, have been taken; and, although the usual symptoms followed, the parties recovered. The last case was the subject of a trial at the Central Criminal Court, in September, 1836. Six drachms of the tincture were administered to a girl, æt. 17; a question here arose respecting the comparative strength of the tincture, and whether half an ounce was sufficient to kill a person. One ounce of the tincture is considered to be equivalent to five and a half grains of the powder; but as the proportion of *cantharidin*, the substance on which the poisonous properties depend, is subject to variation, it is probable that the tincture varies in strength. A case is quoted by Pereira, from Dr. Hosack (*Mat. Med.*, vol. 2, part 2, p. 750), in which it is said six ounces of the tincture were taken by a man without causing dangerous symptoms! This must have been an unusually weak preparation; and probably the insects from which the tincture was made, contained little or no cantharidin. The same writer mentions a case within his own knowledge in which one ounce of the tincture caused serious symptoms. The powder cannot be so readily administered as the tincture, since a large proportion of it floats for a time on any liquid with which it is mixed, and attracts attention by its peculiar appearance.

At the Liverpool Lent Assizes, 1861 (*Reg.* v. *Wilkins*), a man was indicted for administering powdered cantharides to a woman. The prisoner had mixed it with a cup of tea; the prosecutrix took a portion of the tea and suffered from vomiting and other symptoms produced by this substance; she skimmed a quantity of the powder from the tea, on which it floated, and its nature was then easily determined. The prisoner was convicted of the act of administration, but a serious question arose in reference to the intent. The jury found that he had administered the powder with the intent to excite the sexual passion of the woman, for which the new statute had not provided, as this makes the offence to depend only on the intent to injure, aggrieve, or annoy!

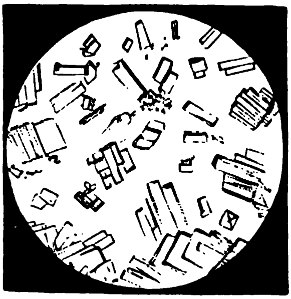
Treatment.—When vomiting exists, this may be promoted by warm demulcent liquids, as thick linseed-tea, or a strong solution of gum arabic; if it does not exist, emetics and castor oil should be given—the object being to dislodge the poison. Demulcent injections may also be used. The inflamed state of the throat may not admit of the application of the stomach-pump. Oil was formerly regarded as an antidote; but it has been found that this is a ready solvent of the active principle, and it is therefore injurious.

Cantharidin—Analysis.—Cantharidin is the vesicating and at the same time the poisonous principle of the insect. It is a white solid crystallizable substance, insoluble in water, but soluble in ether, chloroform, alcohol, fixed oils and caustic alkalies. Although water does not dissolve it in its pure state, it takes it up with other principles from the powdered insect; and thus an aqueous infusion of cantharides is poisonous. Chloroform will separate it from this solution. It is not volatile according to Mr. Procter under 220°. It is powerfully irritant, but requires solution in some menstruum and contact with the skin to produce irritant and vesicating effects. There are no chemical characters by which this principle can be safely identified. We trust to its physiological or *vesicating* properties. The difficulty of extracting *cantharidin* may be conceived, when it is stated, that, according to Thierry's experiments, which are the most perfect, the quantity contained in the fly is only about the 250th part of its weight, so that it would require nearly half an ounce of the powder to yield one grain of cantharidin. The quantity of cantharidin required to produce vesication is extremely small. Robiquet found that the $\frac{1}{100}$th part of a grain, placed on a slip of paper and applied to the edge of the lower lip, caused small blisters in a quarter of an hour.

For the detection and separation of *cantharidin*, it has been recommended to digest the suspected solid or the liquid contents of the stomach (evaporated to an extract) in successive quantities of ether, to concentrate these ethereal solutions by slow evaporation, and then observe whether the concentrated liquid applied to the skin of the lips produces vesication or not—the medical jurist being expected in such cases to make himself the subject of experiment. By this method Barruel discovered cantharides in some chocolate. (*Ann. d'Hyg.*, 1835, vol. 1, p. 455.) For the detection of the powder, M. Poumet recommends that the suspected liquid, mixed with alcohol, should be spread on glass slides or sheets of glass, and allowed to evaporate spontaneously to dryness. The fragments of the shining scales, of a golden green or copper color, will then be seen, on examining by reflected light either one or both surfaces of the glass. (*Ann. d'Hyg.*, Oct. 1842.) Particles of cantharides may be detected in the viscera by this optical method, long after interment. Orfila has detected them after a period of nine months, so that they do not seem to be affected by the decomposition of the body. As the powder is insoluble in water, some portion of it may be obtained by washing and decantation. The sediment may be examined on a glass slide with the microscope.

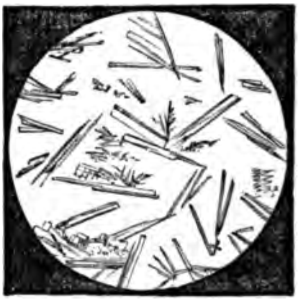
Mr. Tichborne recommends, in place of ether, the use of chloroform for the separation of cantharidin from the tincture or from an alcoholic or aqueous extract of the contents of the stomach. He added four drachms of the tincture, equivalent to three grains of powdered flies, to half a pint of wine; one ounce of chloroform was frequently shaken with this mixture and left in contact with it twenty-four hours. The chloroform was then separated by a funnel and filtered; it was allowed to evaporate spontaneously in a watch-glass. A pellet of lint of the size of half a pea, pulled out, was moistened with a drop of olive oil, and the residue in the watch-glass was taken up by it. It was placed

FIG. 44.



Crystals of cantharidin from a solution in chloroform, magnified 30 diameters.

FIG. 45.



Crystals of cantharidin from a solution in ether, magnified 30 diameters.

upon the arm and covered with gold-beaters' skin. When taken off in three or four hours, the skin was very red, and on wiping it with chloroform, a large vesicle was produced. (*Chem. News*, Feb. 14, 1863, p. 78.) The quantity of cantharidin here detected amounted to only the $\frac{1}{80}$th part of a grain. This mode of operating is certainly preferable to the use of ether, as cantharidin is less soluble in ether than in chloroform. I have ascertained by experiment that the extract obtained from half an ounce of the tincture of cantharides will yield to chloroform a crystallizable principle, having the characters assigned to cantharidin. In practice it will be found advisable to remove the alcohol and concentrate the liquid as much as possible before adding the chloroform, and to employ two measures of chloroform for one measure of the liquid for analysis. Benzole has also been employed successfully by percolation for the separation of cantharidin. It appears to be a powerful solvent of this principle. Sulphide of carbon is used to remove the fatty matters, and the cantharidin is then left in small prismatic crystals. By this process 500 grains of commercial cantharides gave two grains of cantharidin. (*Pharm. Jour.*, February 20, 1875, p. 662.)

Cantharidin is a neutral crystallizable principle contained in the body and elytra of the beetle *Cantharis Vesicatoria.* Illustrations of its crystalline forms obtained by the spontaneous evaporation of its solutions in ether and chloroform are annexed, Figs. 44 and 45.

As ten grains of the powder contain only the $\frac{1}{25}$th part of a grain, it will not be in the power of an analyst to extract cantharidin unless the powder is present in comparatively large quantity. The $\frac{1}{100}$th of a grain of cantharidin dissolved in ether is said to possess vesicating properties.

The evidence of the presence of cantharides, or of their having been taken, is necessary in order to support a criminal charge; for however unambiguous the symptoms produced by this poison may appear to be in its peculiar effects on the generative and urinary organs, a medical jurist should be aware that similar symptoms may proceed from disease. An important case of this kind has been reported by Dr. Hastings (*Med. Gaz.*, vol. 12, p. 431). A young lady was suddenly seized with vomiting, thirst, pain in the loins, strangury, and considerable discharge of blood from the urethra; the generative organs were swollen and painful. She died in four days. She was governess in a family, and there was some suspicion that she had been poisoned with cantharides. The stomach, kidneys, and bladder were found inflamed, and the latter organ contained two ounces of blood. There was no trace of poison, and indeed it was pretty certain from the general evidence that none could have been taken or administered.

CHAPTER LIV.

NOXIOUS ANIMAL FOOD — POISONOUS FISH — MUSSELS — CHEESE — SAUSAGE POISON — DISEASED MEAT — PORK AND BACON — SOURCE OF TAPE-WORM — TRICHINA SPIRALIS — TRICHINOSIS — PUTRESCENT FOOD — POISONED GAME.

NOXIOUS ANIMAL FOOD.

CERTAIN kinds of animal food are found to produce, occasionally, symptoms resembling those of irritant poisoning. In some instances this poisonous effect appears to be due to idiosyncrasy; for only one person out of several may be affected. These cases are of importance to the medical jurist, since they may give rise to unfounded charges of criminal poisoning. In the absence of any demonstrable poison, we must test the question of idiosyncrasy by observing whether more than one person is affected, and whether the same kind of food, given to animals, produces symptoms of poisoning. If, with this latter condition, several persons are affected simultaneously, we cannot refer the effects to idiosyncrasy; they are most probably due to the presence of an animal poison. Among the articles of food which have caused symptoms of irritant poisoning, may be mentioned—

Poisonous Fish.—Some kinds of fish have been long known to produce symptoms resembling those of irritant poisoning, and occasionally neurotic symptoms have been combined. These are avoided as food; but fish reputed wholesome may cause occasionally alarming symptoms and even death. A case of this kind occurred to Mr. Maunder (*Lancet*, July 30, 1864, p. 132), which may serve as an illustration. A man, æt. 35, ate a portion of mackerel and complained of not liking it. For two days subsequently he was not well, but still able to get about. On the third day an eruption made its appearance on the skin, and on the ninth he died. When seen on the morning of this day he was restless and occasionally delirious. The pulse was quick, weak, small, and irregular; the tongue dry and brown, and there was occasional hiccough. The skin was red, excepting that of the face. There was œdema of the upper part of the thighs and the lower part of the abdomen and loins, with vesication. There was a scaling off of the cuticle on the arms, hands, and legs. There appears to have been no vomiting or purging, and no other apparent cause of death.

Mussels.—Of all the varieties of shell-fish, none have so frequently given rise to accidents as the common mussel. The symptoms which it produces are uneasiness and sense of weight in the stomach, sensation of numbness in the extremities; heat, dryness, and constriction in the mouth and throat; thirst, shivering, difficulty of breathing, cramps in the legs, swelling and inflammation of the eyelids, with a profuse secretion of tears, and heat and itching of the skin, followed by an eruption resembling nettle-rash. These *symptoms* are sometimes accompanied by colic, vomiting, and purging. They may occur within ten minutes or a quarter of an hour; but their appearance has been delayed for twenty-four hours. There is generally great exhaustion and debility.

The symptoms here described have proceeded from the eating of not more than ten or twelve mussels. Two cases, reported by Christison, proved fatal, the one in three, and the other in about seven hours. In general, however, especially when there is free vomiting, the patients recover. In the inspection of the two fatal cases above mentioned, no appearance was found to account for death. A case in which two mussels produced, in a boy, aged ten, alarming symptoms, followed by an eruption resembling scarlatina and nettle-rash, will be found elsewhere reported. (*Guy's Hosp. Reports*, Oct. 1850, p. 213.)

R. G., æt. 23, was admitted into Guy's Hospital in March, 1873. He had eaten a quart of mussels and was suffering from symptoms of poisoning. There was vomiting at intervals. The whole of his body was covered with a raised rash which itched intensely, and after a time became scarlet. There was œdematous swelling of the eyelids with injection of the conjunctivæ, twitchings of the muscles of the face, followed by pain in the abdomen and soreness of the limbs. Under the use of emetics and purgatives the man recovered in about a week. (*Guy's Hosp. Reports*, 1874, p. 420.)

In July, 1860, a number of persons living at Tralee were poisoned under the following circumstances. A woman picked up some mussels which she found at the bottom of the basin of a ship-canal. She distributed them among her neighbors, and during the night twenty-one persons who had eaten them, were attacked with symptoms of poisoning. Three children died, and six persons were placed in imminent peril. The rest were soon out of danger. Eight out of the twenty-one attacked were adults. (*Med. Times and Gazette*, July 28, 1860.) In October, 1862, an accident occurred at Liverpool in which a woman died in about four hours, after having eaten some mussels taken from a ship in the docks. Severe pain and vomiting were among the symptoms, which generally resembled those of arsenical poisoning. Several other persons were made seriously ill, but recovered. Although the vessel was not sheathed with copper or yellow metal, it was coated with a green pigment, of which arsenic may have been a constituent. Mr. Paterson, of Liverpool, describes a set of cases, in two of which the symptoms were chiefly those affecting the brain and nervous system. Two persons recovered and two died. On this occasion also the mussels had been collected in the Liverpool docks. (*Lancet*, March 1, 1873, p. 323.) In this year other fatal cases occurred at Falmouth. Three boys after eating mussels became insensible and died within an hour. (*Lancet*, February, 1873, p. 247.) In five years (1863–7) there were eight deaths from mussels recorded.

The poisonous action of mussels can be referred neither to putrefaction nor disease; nor in all cases to idiosyncrasy, since in one instance those mussels only which had been taken from a particular spot were poisonous; all persons who partook of them suffered, and a dog to which some of them were given was killed. From a case which occurred to M. Bouchardat, it would appear that copper is sometimes present, and may be the cause of the poisonous effects. Two women were poisoned by mussels, and he found on analysis sufficient copper in the fish to account for the symptoms of irritation from which they suffered. (*Ann d'Hyg.*, 1837, vol. 1, p. 358.) Copper is not, however, present in all cases, and it is therefore probable that there is in some, if not in all instances, an *animal poison* present in the fish. (See *Ann. d'Hyg.*, 1851, vol. 1, p. 387; vol. 2, p. 108.) *Oysters* and *periwinkles* have occasionally given rise to similar symptoms. *Salmon*, sold in the state of pickled salmon, or even *herrings* salted, may also act as irritants: this may be due to the fish being partially decayed before it is used, or to the noxious effects of the pickle. For some remarks by Dr. Hamilton on the poisonous properties of fish, see the *Pharmaceutical Journal*, January, 1853, p. 344.

Cheese.—The symptoms produced by cheese have been those of irritant poisoning. The nature of the poison is unknown. In some cases the irritant property is due to a putrefied state of the curd, or to the production of a rancid irritant oil. Again, it has been supposed that the poison is occasionally derived from certain vegetables on which the cows had fed. In 1858 a case was referred to me for examination, in which twenty-five persons had suffered from vomiting and purging, more or less violent, owing to their having partaken of cheese. The only articles of food in common were bread, beer, and cheese. The bread and beer were excluded from any suspicion of containing poison. All the persons recovered. On a close examination of the cheese, I found it to be strongly acid; it had an offensive musty smell, and yielded a quantity of acrid oil to ether. It had not been properly pressed, and the casein had undergone chemical changes. The ashes yielded copper and lead, but only in traces. The cheese had acquired irritant properties, not from the presence of any poisonous matter added to it, but from partial decay. There was abundant evidence that cheese from the same dairy had been eaten without causing any injurious symptoms. This negative evidence, however, is quite consistent with one cheese acquiring noxious properties. Dr. Britton, of Driffield, has published a number of cases in which symptoms resembling those of irritant poisoning were caused by cheese. (*Lancet*, March 1, 1873, p. 328.) He traced the illness among different families to cheese procured from the same shop and from the same stock. One cheese only out of a number of the same kind, produced these poisonous effects. The cheese was analyzed, but no poison was found in it. The fatty matter had a rancid smell.

In February, 1865, a set of cases were reported to the Med.-Chir. Soc. of Edinburgh, in which a sample of American cheese had produced alarming symptoms of irritation. They appeared in about three hours after the cheese had been eaten. The persons suffered from severe pain in the stomach—in some cases cramp, with violent vomiting of a greenish fluid, soreness of the throat, and a cold, clammy condition of the skin. All the patients recovered, and in every case recovery was preceded by profuse perspiration. The cheese appeared quite fresh. It had no particular odor, or anything to indicate poisonous properties. (*Ed. Med. Journ.*, 1865, vol. 1, p. 854.) We must not lose sight of the fact that cheese may actually contain poison mixed with it through ignorance. Orpiment or chromate of lead may be used as coloring, and the discovery of such mineral substances would at once account for the irritant effects. (See *Pharmaceutical Journal*, August, 1862, p. 89.) *Annatto*, the coloring matter usually employed to give a yellow or orange color to cheese, is a perfectly innocent extract; but it may be adulterated with colcothar, chrome yellow, orpiment, or other noxious mineral substances, and thus convey poison into cheese.

Milk.—Milk is subject to be affected by disease in the cows, but in this state it acts like an emetic, and produces vomiting. In one instance three children were attacked with violent vomiting after taking milk at their breakfast. I found a quantity of blood mixed with it, and from its appearance there was no doubt that it had been taken from a diseased cow. The parents suspected poison, but there was no poison present.

Sausage Poison.—The symptoms caused by *sausage poison* partake of a narcotico-irritant character; they are very slow in appearing; sometimes two, three, or four days may elapse before they manifest themselves. This poison has been so called because its effects have been chiefly manifested when the flesh has been made into sausages; its effects have been chiefly observed in Germany, where sausages are a staple article of food.

In the *Medical Gazette* for Nov. 1842, there is an account of the cases of three persons who had died from the effects of liver-sausages, which had been made from an apparently healthy pig, slaughtered only a week before. The inspection threw no light on the cause of death. The poisonous property was supposed to depend on a *partial* decomposition of the fatty part of the sausages. It is said that when extremely putrefied they are not poisonous. In a case in which I was consulted, a few slices of a German sausage, evidently of old manufacture, but not putrescent, caused the death of a child, with violent symptoms of irritation of the stomach and bowels. I examined a portion of the sausage; it contained no poisonous matter which admitted of detection. The fatty portions were rancid, and the lean portions very dry. There was no doubt, however, that it had been the cause of the symptoms and death of the child.

Dr. Tripe has published a complete account of the effects produced by sausage poison. (*Brit. and For. Med. Rev.*, Jan. 1860, p. 197.) It appears that in November, 1859, sausages made and sold by a pork-butcher at Kingsland, were eaten, more or less, by sixty-six persons, of whom sixty-four were attacked with violent symptoms of irritation, in from three and a half to thirty-six hours subsequently to the meal. One case only proved fatal, on the seventh day. No symptoms appeared in this man until after the lapse of *six hours*. It seems that he had eaten one of the sausages raw and three cooked. He was attacked with severe vomiting and purging, followed by shivering; there was pain in the abdomen, violent headache, and great prostration. The pulse was feeble and quick, and there was delirium. These symptoms underwent a remission, but he had a relapse, became comatose, and died on the seventh day. Latterly he chiefly complained of pain in the bowels. Dr. Letheby found, on inspection, no signs of inflammation, or of the action of an irritant in the stomach. The small intestines were much inflamed at the lower end, and the gall-bladder was distended. The other organs were healthy. The viscera contained no vegetable or mineral poison. The sausages were made with heifer-beef, pork-fat, sage, and pepper. There was no chemical evidence of anything noxious about them, and an analysis failed to show the presence of poison. There could, however, be no doubt that the sausages had caused the symptoms and death, the food in this case acting as a narcotico-irritant poison. Other persons suffered from burning pain in the throat and stomach, followed by vomiting and purging; then giddiness or confusion in the head, and in some there was delirium. In the man who died, the delirium was well marked, and the eyes were red. In those persons who recovered, the noxious animal matter was probably early thrown off by vomiting and purging. Most of these cases probably arise from the eating of flesh in a diseased state. This view is borne out by a case brought before a metropolitan police magistrate in July, 1874. A part of the meat was seized before it had been chopped up for sausages, and an abscess was found in it. (*Sanitary Record*, July, 1874, p. 54.) The meat would, of course, be equally noxious whether made into sausages or eaten in its ordinary state.

DISEASED MEAT.

Mutton.—The following case was referred to me in December, 1840. Four members of the family of a shepherd were attacked with symptoms resembling those of irritant poisoning after eating a portion of mutton which had been given to them. The father and mother suffered severe pain after the meal, and the latter had an attack of vomiting, and became insensible. Their children, a boy and a girl, were seized with violent vomiting and purging, and the boy died in three hours after the meal. On inspection, the mucous membrane of the stomach presented patches of inflammation, and there was inflammation of the peritoneum. The stomach contained some half-digested food, mixed with blood. No trace of poison was found in the contents, or in the food. It appeared that the mutton was from part of the body of a sheep, which had been killed while affected with the "staggers." The flesh was distributed among several poor familes, including that of the shepherd. It is remarkable that the other families did not suffer. The facts lead to the inference that all parts of a diseased animal may not be injurious. (*Guy's Hosp. Rep.*, 1843, p. 1.)

Veal.—Sir R. Christison met with a case in which *veal* produced similar effects on a family consisting of four adults and ten children. Three hours after the meal they were all seized with pain in the stomach, efforts to vomit, purging, and lividity of the face, succeeded by a state of stupor. One patient died comatose in the course of six hours. The rest, after having been freely purged and made to vomit, recovered, although they were prostrated by exhaustion and collapse.

The calf from which the meat was taken was found dead on the seashore, and there is but little doubt that it was in a state of decay. (*On Poisons*, p. 647.)

Raw or uncooked meat may, when eaten, be a source of tænia or tapeworm. Raw beef sandwiches taken as food apparently gave rise to tapeworm in a case which is reported in the *American Journal of Medical Sciences* for July, 1871, p. 293.

Pork—Bacon.—These common articles of food occasionally produce symptoms so closely resembling those of irritant poisoning, as to be easily mistaken for them. In some cases, the effect appears to be due to idiosyncrasy; but in others it can be explained only by supposing the food to have a directly poisonous action. The noxious effects of pork have been particularly shown by the cases published by the late Dr. Mac Divitt. (*Ed. Med. and Surg. Jour.*, Oct. 1836.) As pork is sometimes salted in leaden vessels, lead may be found in it; but fresh pork has been observed to have a noxious action. In January, 1864, Mr. Kesteven met with a case in which all the members of a family were attacked with symptoms of irritant poisoning after eating a leg of pork. The principal symptoms were nausea, vomiting, griping pains in the abdomen, and purging; but dogs and cats fed upon the meat did not appear to suffer. Other portions of the animal from which the leg was taken were eaten by other families, and no symptoms of poisoning were produced. I examined the food without discovering any trace of the ordinary poisons.

These cases of poisoning by animal food have of late been very fully examined by Mr. Simon and Mr. Gamgee. These gentlemen have traced the injurious effects of pork to a diseased condition of the pig, owing to the animal having been fed on improper food. The term measly pork is now very well known to consist in a diseased condition of the flesh of the animal, in which it is filled with a parasite called cysticercus, which is believed to be the larva of the tapeworm. (See *Med. Times and Gaz.*, 1870, vol. 1, p. 485.) This parasite undergoes full development when in the shape of food it reaches the human intestines. The parasites may not directly kill a person who eats this noxious food, but they favor the development of fatal disease. In reference to the possible ill effects from consuming, in a well-cooked state, the flesh of animals afflicted with *anthrax* or carbuncular disease, evidence is still imperfect, but there is reason to believe that human life may be endangered by it. An opinion has been expressed that boils and perhaps other like affections are caused in human beings by the consumption of diseased meat. According to Mr. Gamgee, at a convict establishment, where diseased cattle are eaten in large quantities, and especially cattle afflicted with lung-diseases, as many as 40 and 50 cases of boils and carbuncles occur in a month among 1500 convicts. (Simon's *Annual Reports*, 1863.) This lends support to the theory that diseased animal food is highly favorable to the production of carbuncular disease.

There is reason to believe that, in spite of every precaution, a large amount of diseased and unwholesome meat is sold to the public, and that of the various kinds of flesh used as food, none is so subject to disease as pork. Some of the changes which it undergoes are of a microscopical character, and are not likely to be noticed. This subject has attracted the attention, not only of the Board of Health in England, but of the Governments of France and the German Empire, with a view to the protection of public health. For a full account of the diseases affecting the flesh of the pig, their mode of production, and the prevention of accidents, I must refer the reader to papers by M. Delpech, in the *Annales d'Hygiène*, 1864, vol. 1, pp. 5, 241. (*De la Ladrerie du porc*, etc.) It has been clearly shown that the parasites found in the flesh of this and other animals are not easily killed by boiling, roasting, or smoking, and that those are liable to suffer the most, who habitually eat the raw or partly cooked flesh.

The flesh of the pig containing cysticercus presents in the cooked state the following appearances: When boiled, it is paler than wholesome meat; it appears dryer in patches, and the muscular fibres are more separated than usual. When these are opened, the parasites are seen in the interstices, appearing as opaque white spots of the size of a hempseed, and presenting much the same aspect as when living. The caudal bladder attached to their bodies disappears when the meat is thoroughly cooked, and the body of the animal then appears isolated in the middle of the muscular tissue. It is friable, and breaks down easily under pressure with a crackling sound, owing to the presence of calcareous matter. In this state it does not appear to be necessarily productive of injury (*Ann. d'Hyg.*, 1864, vol. 1, p. 249), although such food must be regarded as most unwholesome. All the members of a family were seized with vomiting, purging, and syncope after having eaten a dish of pork. A medical man examined the meat, and found it full of cysticercus. A pork-butcher was accused of having sold bad meat, but it was proved to have been some cheap pork bought of a hawker of provisions. (*Ann. d'Hyg.*, 1864, vol. 1, p. 246.) If the cysticercus did not cause the symptoms in this case, the meat had undergone some change sufficient to impart to it irritant properties. These parasites occur in all the fleshy parts of the body. I have seen them in the human heart, as well as in the flesh of animals. My colleague, Mr. Hilton, first showed them to me in the living state, in the muscles of the thigh of a man, in 1834. They are not commonly found in the fatty portions of man and animals, and are less common in sheep and oxen than in pigs.

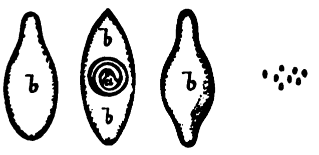
TRICHINOSIS.

The fatal malady arising from the introduction of the trichina spiralis into the human body has attracted much attention in France and Germany; and, among others, Dr. Keller, of Darmstadt, has published some important facts illustrating the symptoms produced, and the mode in which this parasite causes death. Dr. Keller considers that it is a question well worthy of the attention of medical jurists whether many cases of death from suspected irritant poisoning, in which no poisonous matter could be detected in the body, may not have been really due to trichina disease.

The *trichina spiralis* (from θρίξ, a hair) or flesh-worm, is found chiefly in the course of the fibres of all the striped muscles of the trunk and limbs, most frequently on those of the front of the chest, neck, and abdomen. It has also been found in the muscular fibres of the heart and œsophagus or gullet. The parasites appear in the form of very small white ovoid bodies or capsules, perceptible to the eye as white specks, in the midst of the fibres, but only distinctly seen by the aid of the magnifying glass. The trichina or worm is coiled up in the centre of each oval capsule, the greater diameter of which is always parallel to the fibre with which it is closely incorporated. The annexed engraving (Fig. 49) is taken from a preparation in the Museum of Guy's Hospital; it represents a portion of the abdominal muscles of a man, covered with trichinæ *in situ*, and as nearly as possible of the natural size. The other illustrations in Figs. 46, 47, and 48, are from drawings by Dr. Wilks, representing three of the capsules *b b b* magnified, with the trichina coiled up in the centre of one of them.

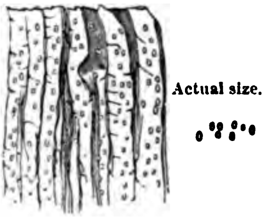
These parasites are frequently so numerous as to give to the red flesh a white or speckled appearance. According to Dr. Keller, as many as 300,000 have been estimated to exist in half a pound of raw meat; and Dr. Pietra Santa affirms that one gramme (about sixteen grains) of diseased meat contained 6000 trichinæ, each having from sixty to eighty embryos. (*Ann. d'Hyg.*, 1864, vol. 1, p. 317.) The actual size of the capsule has been variously stated. From an examination of two sets of specimens in the muscles of the throat and abdomen, I estimated the long diameter to be $\frac{1}{50}$th of an inch, and the short diameter $\frac{1}{100}$th. The worm itself varies from $\frac{1}{20}$th to $\frac{1}{30}$th of an inch in length. The capsules are remarkably uniform in size. They are slowly built up at the expense of the muscular structure by which they are surrounded.

FIG. 46. FIG. 47. FIG. 48.



Magnified view of trichina spiralis in its capsule.
a. The worm coiled.
b. The capsules.

FIG. 49.



Trichina spiralis in the muscular fibres of a man; natural size of the capsules containing the worm.

The history of this parasitical animal has been given by numerous pathologists. (*Hodgkin's Lectures on Morbid Anatomy*, 1836, vol. 1, p. 211.) A full description of its anatomy and habits by Dr. Bristowe and Mr. Rainey will be found in the *Transactions of the Pathological Society* for 1853–4, p. 274. More recent accounts of its influence on health by Dr. Pietra Santa, have been published in the *Ann. d'Hyg.*, 1864, vol. 1, p. 305 ("La Trichina Spiralis"), and by Drs. Schultze and Lücke in Casper's *Vierteljahrsschrift für gerichtliche Medicin*, 1864, No.

1, p. 103, and No. 2, p. 269; also 1874, vol. 1, p. 103. Dr. Lücke's paper is of especial interest, as its title "Die Trichinen vor dem Forum" implies, since it points to the medico-legal bearings of the subject, and the possible danger of confounding the ravages of this worm, with the obscure effects produced in certain forms of chronic irritant poisoning.

From these researches, it is now clearly established that the trichina is a viviparous parasite, which passes the greater part of its existence, in the chrysalis state, in the muscular system of animals, until, by the consumption of the muscle as food, it finds in the stomach and intestines of another warm-blooded animal, a favorable medium for its full development into an intestinal worm. According to Virchow and Zenker, the trichina not only frequently presents itself in the human organism, but this organism is most favorable to its development. The period of incubation of the chrysalis in the stomach and bowels of man or of warm-blooded animals, is from six to eight days; and it there thrives and propagates to an almost incredible extent. Dr. Keller states that in three or four days the female produces 100 or more young ones, which begin on the sixth day to leave the parent animal; and he estimates that in a few days after the ingestion of half a pound of meat, the stomach and intestinal canal of a person may contain thirty millions of these minute worms! M. Herbst found the muscles of two dogs which had been fed upon parts of a badger containing the worms, to be loaded with trichinæ. When once introduced into the stomach and intestines, the worms leave their capsules, become free and produce young, which migrate through the walls of the intestines into the muscles; there they became encysted, and are ultimately found appropriating and destroying the muscular substance to a greater or less extent. After a long residence in the muscle, they appear to acquire calcareous cysts, like minute shells. The sudden liberation of a large number of these worms causes irritation and inflammation in the bowels, attended with peculiar symptoms, resembling in some respects those of chronic irritant poisoning.

It is worthy of note that trichinæ are more frequently found in pork and articles of food derived from it, than in any other kind of meat. Measly pork appears to be sometimes of a trichinous character. Further, the vitality of the parasites is not destroyed unless the meat or other substance in which they are located, has been subject to a temperature equal to that of boiling water for a sufficient time to insure that every particle has been exposed to this degree of heat. Salting and smoking, or partial cooking is not sufficient to destroy the worms in all parts of the food, and they have even been found living in putrefied meat. This may serve to account in some cases for the serious symptoms which have followed the use of pork as food, also of bacon, sausages, and German sausages, which are generally made of raw ham. It is stated on good authority that this parasitic disease does not attack sheep, oxen, or horses, and that beef is the safest of all descriptions of food, as no parasites have ever been discovered in it. They have not been found in the blood of animals whose muscles are liable to their attacks.

Symptoms and Appearances.—The symptoms produced by the use of such food are, in the first stage, those of intestinal irritation, loss of appetite, sickness, pain, general weakness of the limbs, with diarrhœa, swelling of the eyelids and of the joints, profuse clammy perspiration, and a wasting fever, sometimes of a typhoid character. Death is either the result of paralysis (from destruction of the muscular fibres), or of peritonitis and irritative fever. During the perforation of the coats of the intestines by these worms, the mucous membrane becomes irritated and inflamed; pus is formed on its surface, and bloody evacuations are sometimes passed. No case is known in which trichinosis, after having once declared itself, was arrested by medical treatment.

The noxious effects of this food on human beings are well illustrated by a series of cases which occurred at Hettstädt, in the Hartz Mountains, in 1863. (*British Med. Journal*, Jan. 16, 1864, p. 75.) One hundred and three persons partook of smoked sausages made from a pig affected, as it turned out, with trichinous disease. The sausages were fried, and served for dinner in the usual way. On the following day, several persons who had partaken of this food were attacked with severe pain in the bowels, purging, loss of appetite, great prostration of strength, and fever. The number of persons attacked rapidly increased; symptoms of peritonitis and pneumonia manifested themselves, and these were followed by paralysis of the intercostal muscles, and of the muscles in front of the neck. Eighty-three persons died from the effects of this noxious food, and the remainder were seriously injured in health. The remnants of sausage and of pork not eaten at this festival were examined, and were found to be literally swarming with encysted trichinæ. (See Casper's *Vierteljahrsschrift*, April, 1864, p. 286; also *Ann. d'Hyg.*, Oct. 1863, p. 471, and April, 1864, p. 321.)

There were two outbreaks of this disease in Massachusetts in 1870. A family consisting of six persons partook of a dinner of fried fresh pork on February 8th. A portion of the meat was underdone, and the members of the family who ate the red and imperfectly cooked parts suffered most. Three escaped entirely, and three were affected on the 13th with symptoms of trichina disease. They all suffered from a long and painful illness, and one died on March 12. Portions of the muscles taken from the dead body were swarming with living trichinæ. No portion of the pork could be obtained for examination, nor any history of the pig from which it was taken. In the other set of cases the disease was communicated to a family through a smoked ham, from a pig which had exhibited no sign of the disease during its life. The family consisted of father, mother, and six children. The two youngest ate none of it. The father ate a portion, slightly cooked, and the rest of the family consumed it raw, cut in thin slices like smoked beef. The first symptoms were those of an ordinary cold, followed by fever, loss of appetite, swelling of the face and eyelids, great muscular pains, contraction of the flexor muscles, and other symptoms. None of these cases proved fatal. The portions of the ham which remained were filled with living trichinæ. Mere smoking does not destroy them, but if the pork is well boiled or thoroughly cooked throughout its whole substance, it is rendered innoxious. (*Lancet*, 1871, vol. 1, p. 515.) In the same volume, p. 710, a fatal case is mentioned as resulting from pork. Many persons were taken ill; but, with one exception, they recovered. In 1866, Dr. Smith, of Iowa, met with a case in which nine persons were poisoned by eating trichinous pork. The symptoms occurred four or five days after they had eaten some raw smoked ham. Five of the patients died. Trichinæ were found in great number in the muscles.

Although little has been heard of trichinous disease in England, it appears to be prevalent in Germany. Among the official notices published by the German government, is one which points out the danger arising from the use of pork in a diseased state, and warning those who sell it to the public of the penalties which they thereby incur. (Casper's *Vierteljahrsschrift*, July, 1863, p. 177.) In 1862, thirty-eight persons in Kalbe suffered severely from the use of such food, and of these eight died from the effects.

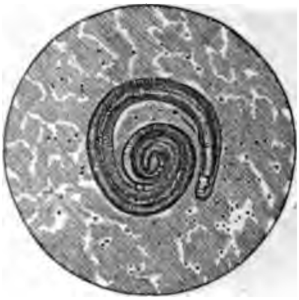
In Hasselbusch, early in March, 1874, a lady, her servant, and eight other persons were infected by eating a small quantity of raw pork. Several children in Rapendorf were attacked at the same time from eating a portion of the same pork. In Lissa, five persons were attacked who had partaken of a ham, which was found, on examination, to be full of trichinæ. (*Brit. Med. Jour.*, April 11th, 1874, p. 494.)

In July, 1874, several patients were admitted into the Berlin Hospital, suffering from trichinæ, in consequence of eating raw ham. One case proved fatal. The *British Medical Journal*, August 8th, 1874, reports that many persons had been attacked with the disease owing to the use of raw pork. In one out of nine villages affected, there were no fewer than forty-three cases.

Dr. Schultze refers to other groups of cases, which, however, do not appear to have been attended with great fatality. In Magdeburg, Neustadt, and Buckau, over a period of five years (1858–62), from three to four hundred cases of illness were traceable to this cause. ("Die Trichinen Krankheit," Casper's *Vierteljahrsschrift*, April, 1864, p. 278.) In Burg more than fifty persons suffered, and eleven died. Other fatal cases have been reported. For these I must refer the reader to Dr. Schultze's paper. In February, 1864, a whole family was poisoned at New York, and one member died from eating part of a ham, which, on microscopic examination, was found to be full of trichinæ. Death was referred, by the medical attendant, to this cause. There were two outbreaks of this disease in Massachusetts in 1870. Out of six persons who ate the noxious food, one died. (*Lancet*, 1871, vol. 1, pp. 515 and 710.) In May, 1865, Senator Dittmere, of Lubeck, and his family, consisting of seven persons, were seized with symptoms resembling irritant poisoning, some time after eating a ham which had been smoked but not cooked. On examination it was found to be loaded with trichinæ. Four out of those attacked died. (*Lancet*, May 27, 1865, p. 562.)

It is probable that some unexplained cases of illness or death from irritation of the stomach and bowels, simulating chronic irritant poisoning, may have been the result of eating trichinous food. Medical men have been unable to group the symptoms under any known form of disease, while the marks of irritation in the mucous membrane of the bowels have given strength to the supposition that poison must have been taken by the deceased, although chemical analysis had failed to show the presence of any ordinary poison in the fluids and solids of the body. In the course of many years' practice, I have met with several cases of this description, and there has been sometimes manifested a disposition to doubt the accuracy of chemical analysis. Dr. Lücke has related a series of fatal cases which occurred in 1845, attributed at the time to poison, which, as he suggests, were most probably caused by the use of trichinous food. (Casper's *Vierteljahrsschrift*, Jan. 1864, p. 102.)

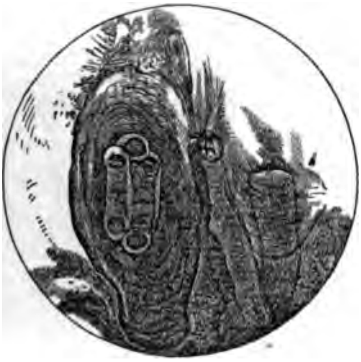
FIG. 50.



Single trichina spiralis from human muscle, magnified 150 diameters.

This was taken from a case which proved fatal in six weeks. The worm was found to be 1-20th of an inch in length.

FIG. 51.



Trichina spiralis encysted in pork.

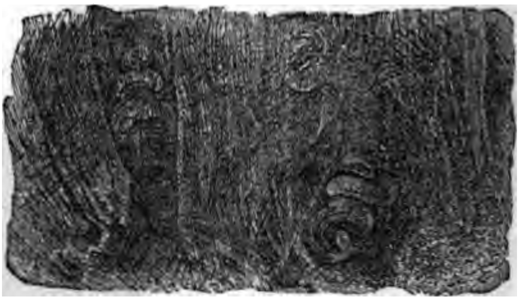
This engraving represents the completely encysted worm in horizontal sections, showing six stumps, which proves that the worm lies in three convolutions when it has attained its full size.

As means of distinction from irritant poisoning may be pointed out the long time which commonly elapses between the taking of the food and the commencement of the symptoms. The pain, vomiting, and purging are comparatively slight; the pain is in the bowels rather than in the stomach. Peritonitis, pneumonia, and fever are not commonly results of the action of irritant poisons, while these diseases appear in cases of trichinosis. The absence of ordinary poison in the food, in the urine, and the evacuations at any stage, may also be taken as conclusive evidence against irritant poisoning in its usual form.

In suspected cases, a new method of research must be added to those already in use. If any of the food can be obtained, this must be examined for the parasite by the aid of the microscope. If the case proves fatal, the voluntary muscles of the deceased must undergo a similar examination. In the *Canada Medical Journal* for 1870–1, Dr. J. B. Edwards has published a full account of the best methods of detecting trichinæ in the flesh of man and animals. He has furnished me with some excellent photographs of the worms *in situ* in human and animal muscle, of which engravings are annexed.

In some cases which occurred at Montreal the cause of the symptoms was at first obscure, but Dr. Edwards not only found trichinæ in a slice of ham which had been eaten, but in the muscles of two of the patients who recovered. He removed about five grains of muscular tissue from the gastrocnemius muscle, and twelve grains of the tibialis posticus. In these two portions of muscle, about forty worms were found. (*Op. cit.*, p. 517. See also Dr. Thudichum's *Report to the Privy Council for* 1864–5.) The annexed engraving (Fig. 52) is of great interest.

FIG. 52.



Trichina spiralis in human muscle, magnified 150 diameters.

It represents a section of human muscle from one of the fatal cases at Hamilton. Two generations of worms are visible in this muscle, those in the spiral form being a young generation marching past, while the upper curl on the right is the only portion in focus of a large worm which lies closely curled up and is slightly encysted.

PUTRESCENT FOOD.

The effects of disease on animal food must not be confounded with those which result from decay or putrefaction. The flesh of the most healthy animal is rendered unfit for food when it has passed into a putrescent state. It is not merely unwholesome, but highly irritant, causing rapidly vomiting, purging, pain, and other symptoms of a severe kind. Fortunately these symptoms lead at once to the expulsion of the noxious food from the body, and the person then recovers; the young, the old, the infirm may, however, be so prostrated by vomiting and purging, that they may sink from exhaustion. Animal matter in a state of partial decay, or in the transition stage of putrefaction, must be regarded as of a poisonous nature. Much of the cheap butcher's meat sold to the poor is in this condition, and is quite unfit for human food. In one year 114,000 pounds of diseased, and 76,000 pounds of putrid meat were seized and condemned in the City of London alone. In January, 1851, the family of a surgeon near London were all affected with symptoms resembling irritant poisoning, after having partaken of a hare which had been stewed in a clean earthen vessel. The surgeon informed me that on the second day his wife was seized with vomiting and purging, giddiness, heat in the throat, and general numbness, with inflamed eyes. Other members of the family vomited, and in the course of a few days the symptoms disappeared. I examined the vomited matter, and found it to consist of portions of the hare, partially digested, but in a state of putrefaction, so that there was abundant evidence of sulphuretted hydrogen in the liquid. There was no mineral poison of any kind, although the symptoms, it will be observed, were rather like those occasioned by arsenic. It had been remarked by the family that a silver spoon, which was used for serving out this unwholesome food, was turned of a brown color, no doubt from the chemical action of sulphuretted hydrogen; and this may be taken as a good domestic test of the putrefied condition of such food. Nature generally applies an appropriate remedy in these cases: the food itself produces copious vomiting and purging.

Cases of this kind must be distinguished from those in which *poisoned game* is sold to the public. The game may be quite free from putrefaction, but rendered noxious by the poisoned grain which may have caused death. Some years since it was a common practice to steep grain in a solution of arsenic, previous to sowing, and pheasants, partridges, and other birds were accidentally destroyed by eating the poisoned grain. In some instances, grouse and other game were maliciously destroyed by the laying of corn, saturated with arsenic or other poisons, in the localities where the birds abound. There is no law to prevent the sale of poisoned game by poulterers, and there is no precaution which can be taken by the purchasers, except by observing whether the birds have or have not been shot. (See on this subject a letter by the late Dr. Fuller, *Med. Gaz.*, vol. 42, p. 1036.)

Mr. Taylor, of Romsey, has directed attention to the serious symptoms produced by *Canadian partridges.* A lady who had partaken of this food was, in about two hours and a half, attacked with the following symptoms: She had sickness, and became insensible; the skin was cold, and no pulse could be felt. She was in a hopeless state for some hours, and only slowly recovered. The birds were quite fresh, having been packed in ice. In another case there were similar symptoms, with constriction of the throat and great pain. Animals were made ill by this food. It was believed that, in these cases, the birds had not been killed by poison, but that their flesh had been rendered poisonous by some vegetable which they had eaten. It is stated that in some parts of Australia the mutton is rendered poisonous by reason of the sheep feeding on poisonous plants. (*Med. Times and Gaz.*, 1871, vol. 1, p. 728.)

NEUROTIC POISONS.

CEREBRAL OR NARCOTIC POISONS.

CHAPTER LV.

ACTION OF NEUROTIC POISONS—OPIUM AND LAUDANUM—SYMPTOMS—PERIOD OF COMMENCEMENT—DEATH AFTER REMISSION—CHRONIC POISONING—OPIUM EATING—EFFECTS OF EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—DEATH FROM SMALL AND RECOVERY FROM LARGE DOSES—FATAL EFFECTS ON INFANTS—PERIOD AT WHICH DEATH TAKES PLACE—TREATMENT.

THE effects produced by the neurotic class of poison on the system have been already described (page 75). They are chiefly referable to disorder of the brain, spinal marrow, and nervous system. The most prominent symptoms are headache, giddiness, paralysis, insensibility, and convulsions. The brain is the organ upon which a cerebral or narcotic poison primarily acts; but in some cases, by the occurrence of convulsions, there is an indication of a remote effect upon the spinal marrow. The distinction between irritant and neurotic poisons is well marked, so far as symptoms are concerned. Neurotic poisons are destitute of any acrid or corrosive properties; they have no local chemical action on the mouth and fauces, and they rarely give rise to vomiting or purging. When they prove fatal, they do not commonly leave any well-marked appearances in the stomach or bowels like the irritants. There is sometimes a fulness of the vessels of the brain and its membranes; but effusion of blood is rarely observed. It is usually said that they do not produce any redness of the mucous membrane of the stomach or intestines; this appearance has, however, been met with on several occasions in poisoning with alcohol and prussic acid. Opium does not cause inflammation of these organs, and when this condition has been found, it may probably be ascribed to the action of alcohol, in which the opium has been dissolved.

Alkaloids and their Salts.—The alkaloids are chiefly derived from the neurotic class of poisons. They are crystalline substances, for the most part colorless when pure, not very soluble in water, but readily dissolved by alcohol, ether, chloroform, and benzole; the three latter liquids having the power of removing them from water when not combined with acids. Hydrate of chloral is also a solvent for many of them. In the acid state their solutions readily undergo the process of dialysis, and many of them may thus be obtained in a condition fitted for testing. Most of them are solid, and are decomposed when heated; two, namely, conia and nicotina, are liquid and volatile. They also possess powerful and peculiar odors; but the greater number of these bodies are inodorous. They have generally a bitter taste, and impart this when in small quantity to organic liquids. Their crystalline forms are different, and some may be thus recognized. They have but a feeble alkaline reaction, and they readily combine with acids for the most part to form soluble salts. Chloroform is an important agent in separating some of these bodies from organic liquids, and it may be well to point out here that this liquid dissolves them in different proportions. According to Pettenkofer, 100 parts by weight of chloroform dissolve of morphia 0.57; of cinchonia, 4.31; of strychnia, 20.16; of narcotin, 37.17; of atropia, 51.49; of brucia, 56.79; of quinia, 57.47; and of veratria, 58.49. Morphia is eminently distinguished from the other alkaloids by its slight solubility in this liquid. It is nearly equally insoluble in ether and benzole.

Certain test solutions have been proposed for determining the presence of an alkaloidal salt when dissolved in water. 1. The *chloriodide of potassium* and *mercury*, made by dissolving 16 grains of corrosive sublimate and 60 grains of iodide of potassium in four ounces of water. This throws down a whitish precipitate in a solution of an alkaloidal salt even when the quantity is very small. The precipitate consists of an insoluble compound of an hydriodate of the alkaloid with iodide of mercury. The test does not precipitate ammonia, but gives yellow peroxide of mercury with potash or soda. It also precipitates albuminous substances; hence the liquid should be first boiled in order to remove these. The precipitation is prevented if a large excess of alcohol or acetic acid is present. When proper precautions are taken, a negative action of this test indicates the *absence* of an alkaloid or alkaloidal salt, and this is often a matter of medico-legal importance.

The limits of this test-liquid for the different alkaloids are represented by the following proportions according to M. Bauer. Distinct evidence is obtained with a solution containing one part of the alkaloid dissolved in the proportions of water which follow: of morphia and nicotina 2500; of narcotin 5000; of atropia 7000; of conia 8000; of strychnia 15,000; and of brucia and quinia 50,000. The presence of any of these alkaloids may therefore be detected by this test, even when the solutions are largely diluted. (*Pharm. Jour.*, Nov. 21, 1874, p. 402.) The test is sometimes made by simply precipitating a strong solution of corrosive sublimate with iodide of potassium, and continuing to add the latter until the precipitate of red iodide of mercury is just redissolved. The presence of one grain of strychnia dissolved by an acid and diffused through six ounces of water, is revealed by this test.

2. The ioduretted iodide of potassium suggested by Herapath and Bouchardat. This is made by dissolving 24 grains of iodide of potassium and 8 grains of iodine in one ounce of water. This solution gives a deep brown precipitate, a compound of iodine and the alkaloid in a solution of an alkaloidal salt, even when present in very minute quan-

tity. It does not precipitate the alkalies, but it may form an explosive compound with ammonia if present in an organic liquid. Before applying either of these tests the organic liquid supposed to contain the alkaloidal salt should be filtered, and if viscid and much colored a part of it should be dialyzed (see p. 155).

The first group of neurotic poisons (p. 73) includes the substances which primarily and specially affect the brain—CEREBRAL POISONS—of these opium and prussic acid may be taken as types.

OPIUM—LAUDANUM.

General Remarks.—OPIUM is a solid vegetable extract—the concrete juice of the unripe capsules of the *Papaver somniferum*. It is sometimes taken in this state as a poison, but more commonly in solution in alcohol under the form of tincture, or as it is popularly called, LAUDANUM. Its poisonous properties are principally due to the presence of an alkaloid, *Morphia*, which exists in it in a state of combination with a vegetable acid, the *Meconic*. Opium contains a proportion of morphia—varying from two per cent. in the Bengal variety to about nine per cent. in certain varieties obtained from the East Indies. According to some chemists, good opium will yield from ten to thirteen per cent. of morphia. The Turkey opium contains on an average about six per cent. according to the analyses of Mulder; but the best kinds of Smyrna opium contain thirteen per cent. (Pereira, *Mat. Med.*, vol. 2, pt. 2, p. 606.) This difference in the quantity of morphia contained in the drug, may sometimes account for certain differences observed in the effects produced by particular doses. One fluid ounce of the tincture of the British Pharmacopœia, contains the soluble matter of thirty-three grains of opium. This is in about the proportion of fourteen and a half minims of the tincture to one grain of dry opium (Garrod). The strength of the tincture as it is procured from different druggists varies greatly. (See Pereira, *Mat. Med.*, vol. 2, pt. 2, p. 647; also *Lancet*, March 12, 1853, p. 251; and *Pharm. Jour.*, 1851, p. 250.)

There is no form of poisoning so frequent as that by opium and its various preparations. In two years, there were one hundred and ninety-six fatal cases in England and Wales, forming nearly two-thirds of all the cases of poisoning. One-seventh of these were cases of children poisoned by overdoses of opium or its compounds, and most of the others were the result of suicide or accident. It is calculated that *three-fourths* of all the deaths from opium, take place among children *under five years of age!* This, however, forms but a small proportion of the actual number of cases; since there is no kind of poisoning wherein recoveries are so frequent. The total number of deaths in five years, 1863–7, from opium and its preparations, amounted to 540, of which four-fifths were among infants and children.

The poisonous salt of opium, meconate of morphia, is soluble in water, alcohol, and diluted acids. The aqueous and alcoholic solutions have an acid reaction. The solutions have a peculiar taste and odor, and by the latter, the presence of opium may be in general recognized. The taste is bitter.

Extract of opium may be regarded as a pure form of the drug. It contains a larger proportion of the poisonous alkaloid morphia. Three grains of extract are equal to about five grains of crude opium. It is employed in medicine in doses of from one-quarter of a grain to three or four grains. The alcoholic solution, under the name of *laudanum*, is sold to the public, in quantities of from half a drachm to two drachms, for twopence—from two drachms to four drachms, for fourpence—exceeding this quantity, eightpence and one shilling per ounce. It is very often sold by ignorant drug-dealers for tincture of rhubarb or black draught.

Symptoms.—The symptoms which manifest themselves when a large dose of opium or of laudanum has been taken, are in general of a uniform character. They consist in giddiness, drowsiness, a strong tendency to sleep, stupor, succeeded by perfect insensibility, the person lying motionless, with the eyes closed as if in a sound sleep. In this stage he may be easily roused by a loud noise, and made to answer a question; but he speedily relapses into stupor. In a later stage, when coma has supervened with stertorous breathing, it will be difficult, if not impossible, to rouse him. The pulse is at first small, quick, and irregular, the respiration hurried; but when the individual becomes comatose, the breathing is slow and stertorous; the pulse slow and full. The skin is occasionally cold and pallid—sometimes livid; at other times warm and bathed in perspiration. The pupils in the early stage are contracted, in the later stage, and when progressing to a fatal termination, they may be found dilated. In a case referred to me in 1846, one pupil was contracted and the other dilated. They are commonly insensible to light. The expression of the countenance is placid, pale, and ghastly; the eyes are heavy, and the lips are livid. Sometimes there is vomiting, or even purging; and if vomiting take place freely before stupor sets in, there is great hope of recovery. This symptom is chiefly observed when a large dose of opium has been taken; and it may be perhaps ascribed to the mechanical effect of the poison on the stomach. The peculiar odor of opium is occasionally perceptible in the breath. In cases likely to prove fatal, the muscles of the limbs feel flabby and relaxed, the lower jaw drops, the pulse is feeble and scarcely perceptible, the sphincters are in a state of relaxation, the pupils are unaffected by light, the temperature of the body is low, there is a loud mucous rattle in breathing, and convulsions are sometimes observed before death; these are more commonly met with in children than in adults. One of the marked effects of this poison is to suspend all the secretions except that of the skin. During the lethargic state, the skin, although cold, is often copiously bathed in perspiration. It is a question yet to be determined, whether this may not be the medium by which the poison is principally eliminated. Nausea and vomiting, with headache, loss of appetite, and lassitude, often follow on recovery.

The contracted state of the pupils has been hitherto considered to furnish a valuable distinctive sign of poisoning by opium or the salts of morphia. In relying upon it, it is necessary to bear in mind the fact pointed out by Dr. Wilks, that, in apoplexy which is seated in the pons varolii, the pupils are also contracted. He describes two cases of this form of apoplexy which were mistaken for poisoning by opium in consequence of this state of the pupils. (*Med. Times and Gaz.*, 1863, vol. 1, p. 214.)

The symptoms usually *commence* in from *half an hour* to *an hour* after the poison has been swallowed. Sometimes they come on in a few minutes, especially in children, and at other times their appearance is protracted for a long period. In a case reported by Dr. Skae, the person was found totally insensible in *fifteen minutes.* As we might expect, from the facts connected with the absorption of poisons, when the drug is taken in the *solid* state, the symptoms are commonly more slow in appearing than when it is *dissolved* in water or alcohol. In a case which occurred at Liverpool, in August, 1863, communicated to me by Dr. Edwards, a lady took, on an empty stomach, a large dose (supposed to have been one ounce and a half) of laudanum. No symptoms of narcotic poisoning appeared for four hours and a half, and life was protracted for twenty-two hours. In a case which occurred to Dr. Gibb, *nine hours* elapsed before the usual symptoms were manifested. (*Lancet*, July 25, 1857.)

The period at which the cerebral symptoms commence is a question of some importance in relation to the retention of power on the part of a person to perform certain acts indicative of volition and locomotion after having swallowed a large dose of this poison. Thus the narcotic effects may not come on until the deceased has had ample time to attempt suicide in some other way. In March, 1843, a gentleman committed suicide at Hammersmith; he was found suspended by a silk handkerchief; but it was shown that he had previously swallowed a large dose of laudanum. There was no doubt that he had died from hanging. In general, it must be allowed as at least possible that a person who has taken a sufficient quantity of this poison to prove fatal, may move about and perform many acts for one or two hours afterwards, but this power ceases when the cerebral symptoms commence. In a fatal case of poisoning by opium which occurred to Dr. Skae, he ascertained that the person rose from his bed, and had moved about his room, at least two and probably *three hours* after having taken poison. (*Ed. Med. and Surg. Journal*, July, 1840.) In another instance, in which the quantity taken was probably half an ounce, but enough to destroy life, the person was able to converse cheerfully and readily with a neighbor *two hours* after she had swallowed the poison.

It has been frequently observed, in cases of poisoning by this drug, that a person has recovered from the first symptoms, and has then had a relapse, and died. There is some medico-legal interest connected with this state, which has been called secondary asphyxia from opium, although there appears to be no good reason for giving to it this name. In December, 1843, a gentleman swallowed a quantity of laudanum, and was found laboring under the usual symptoms. The greater part of the poison was removed from the stomach by the pump; and he so far recovered from his insensibility as to be able to enter into conversation with his medical attendant; but a relapse took place, and he died the following night. The case of the Hon. Mrs. Anson (Jan. 1859) furnishes another illustration of this singular condition. This lady swallowed, while fasting, an ounce and a half of laudanum by mistake. In a quarter of an hour emetics were given, but she did not vomit for half an hour; and she was not treated medically for two hours and a half. The matter then drawn from the stomach had no smell of laudanum. She was quite unconscious, and had lost the power of swallowing. After remaining in this comatose state for upwards of nine hours, the patient revived, her face became natural, the pulse steady, the power of swallowing returned, she was able to recognize her daughters, and, in a thick voice, to give an account of the mistake she had made. This state lasted about five minutes; the torpor then returned, she again sank into profound coma, and died in fourteen hours after the poison had been taken. It is not improbable that, in these cases, death may be occasioned by the accumulation of the poison, carried by the absorbents into the blood: *i. e.*, the morphia may be more rapidly carried into the system than it is eliminated from it. A remarkable case, illustrative of this *remittent* form of poisoning by opium has been published by Mr. Kirby. (*Dubl. Med. Press*, December 24, 1845, p. 406.)

In compound poisoning, where laudanum is one of the ingredients, it may be expected that symptoms of narcotic poisoning will first show themselves. In a case which occurred to Mr. Beatty, a woman, æt. 22, swallowed a packet of Battle's Vermin Killer, and immediately afterwards two drachms of laudanum, and then half a drachm of red precipitate. In three hours she was found to be suffering from narcotic poisoning alone. None of the effects of strychnia had been produced, and there was no irritation from the mercurial poison. Emetics were given, and the stomach-pump used. By this a quantity of Battle's powder and red precipitate were removed from the stomach. Albumen was given, and the woman slowly recovered. (*Lancet*, 1871, vol. 2, p. 907.) It was inferred that the opium had here prevented the action of strychnia. It would have been more satisfactory if the powder drawn from the stomach had been examined and strychnia found in it. There were no symptoms of poisoning by strychnia at any time, although three hours had elapsed before remedies could be applied.

Chronic Poisoning with Opium—Opium-eating.—When opium is taken for a long period in small doses which are gradually increased, its effects are very different. It is this state which we witness in those persons who are addicted to opium-eating. There is no poison which appears to be so much under the influence of habit as opium (p. 66). Böcker met with several opium-eaters, one of them a physician, who took daily thirty grains of solid opium. The English opium-eater (De Quincey) took at one time, for a daily quantity, nine ounces of laudanum. The injurious effects of the drug thus taken for a long period of time, have already given rise to an important question in law relative to life insurance (case of the *Earl of Mar*, 1828); and it will be, therefore, proper to state those facts which have been ascertained with respect to the influence of this practice on health. One of the best descriptions of the effects of opium-eating is that given by Dr. Oppenheim in his account of the state of medicine in Turkey. He says, "The habitual opium-eater is readily recognized by his appearance. A total attenuation of body—a withered yellow countenance—a lame gait—a bending of the spine, frequently to such a degree as to cause the body to assume a circular form—and glassy deep-sunken eyes, betray him at the first glance. The digestive organs are in the highest degree disturbed; the sufferer eats scarcely anything, and has hardly one evacuation in a week; his mental and bodily powers are destroyed. As the habit becomes more confirmed, his strength continues decreasing, the craving for the drug becomes greater; and in order to produce the desired effect, the dose must be constantly augmented. After long indulgence, the opium-eater is subject to neuralgic pains, to which opium itself brings no relief. These persons seldom attain the age of forty, if they have begun to use opium early." This description of the effects is exactly what we should expect from physiological and pathological reasoning. A remarkable instance of chronic poisoning by opium, which occurred to Dr. Myers, will be found in the *Edinburgh Med. Jour.*, 1855–6, vol. 1, p. 357.

The chronic form of poisoning, as witnessed among children in the factory districts, has been described by the late Mr. Grainger. It appears that laudanum, and other preparations of opium, are given to children in gradually increased doses, until the child will bear from fifteen to twenty drops of laudanum at a time. The child becomes pale and wan, with a peculiar sharpness of the features, and rapidly wastes away. The majority of these children die before reaching the age of two years.

Effects of External Application.—Opium, and all preparations containing morphia, have the property of affecting the body through the skin. Some remarks on this subject have been made elsewhere. (See *ante*, p. 25.) Excepting in cases of idiosyncrasy, or where a large quantity of the drug is applied to an abraded surface, they are not likely to produce fatal effects by this mode of introduction into the system. The application of opium, in any form, or of morphia, to an abraded or diseased surface, is liable to give rise to all the results of narcotic poisoning. A young woman, laboring under scirrhus of the uterus, and suffering from vomiting and pain in the stomach, was ordered to apply to the pit of the stomach, from which the skin had been previously removed by a blister, the $\frac{1}{23}$d part of a grain of the muriate of morphia. The same dose was repeated by the endermic process the following morning. Some time afterwards, the woman fell into a state of complete narcotism. She suffered from pain in the head, stupor, ringing in the ears, dizziness, and incoherency, a hot and dry skin, and a strong and frequent pulse. Among the symptoms was one somewhat remarkable, namely, that she saw only the half of surrounding objects: for instance, in the case of a person standing before her, she could see only the right or left half of the body. The cerebral congestion was followed by convulsions. The symptoms gradually abated, but it was three weeks before vision and speech were perfectly restored. (*Oesterreichische Medicinische Wochenschrift*, April, 1845.) Four-tenths of a grain of the acetate of morphia applied to a blistered surface have been known to cause dimness of vision and delirium. The dose commonly recommended for application, endermically, is from one to two grains.

In a case communicated to me a few years since, thirty grains of morphia in powder were applied by an ignorant quack to the ulcerated breasts of a woman. She soon became comatose, and died in ten hours.

Opiate preparations introduced into a wound, or as injections into the *rectum*, may also produce fatal effects. Orfila relates the case of a man who died from the effects of an injection containing thirty grains of opium. (*Op. cit.*, vol. 2, p. 225.) A child has been killed by ten grains of the sulphate of morphia, given in the form of an enema, by mistake for sulphate of quinine. (*Med. Gaz.*, vol. 4, p. 220.)

Appearances after Death.—In a case of poisoning by opium, which proved fatal in fifteen hours, examined at Guy's Hospital, the vessels of the head were found unusually congested throughout. On the surface of the forepart of the left hemisphere there was ecchymosis, apparently produced by the effusion of a few drops of blood. There were numerous bloody points on the cut surface of the brain; there was no serum collected in the ventricles. The stomach was quite healthy. Fluidity of the blood is mentioned as a common appearance in poisoning by opium. There is also engorgement of the lungs—most frequently, according to Sir R. Christison, in those cases which have been preceded by convulsions. (*Op. cit.*, p. 732.) Among the external appearances, there is often great lividity of the skin. Extravasation of blood on the brain is rarely seen; serous effusion in the ventricles, or between the membranes, is much more common. The stomach is so seldom found otherwise than in a healthy state, that the redness, said to have been occasionally seen, may have been due to accidental causes. In a case in which a woman, æt. fifty-six, died in twenty-two hours after taking half an ounce of tincture of opium, the following appearances were found twenty-four hours after death. The brain was congested throughout, the blood in the sinuses fluid, and there was about an ounce of serum effused between the membranes and in the ventricles. The lungs were emphysematous in places, and the liver was rather enlarged and congested. The intestines were nearly empty, had patches of congestion, with bloody mucus on the lining membrane. The stomach presented at the larger end some redness of the mucous membrane, but apparently of a pseudo-morbid character. There was no smell of opium in the contents, nor could the least trace of meconic acid or morphia be detected in them.

In a case of poisoning by a large dose of tincture of opium, Dr. Sharkey found the following appearances twelve hours after death. The body was warm and rigid; the stomach healthy, containing a quantity of a gruel-like fluid, without any *smell* of opium. The intestinal canal, and the viscera of the abdomen, were healthy. The veins of the scalp, as well as of the dura mater and sinuses, were gorged with blood; but there was no effusion in any part of the brain. The contents of the stomach yielded no trace of morphia or meconic acid; but there was no doubt that death had been caused by opium, taken the previous night. (*Med. Gaz.*, vol. 37, p. 235.) In another case of death from a small dose of extract of opium, this gentleman found great lividity of the skin of the neck. The scalp, superficial veins, and sinuses of the brain contained a large quantity of blood; and there was effused blood, both fluid and coagulated, around the upper part of the spinal marrow. There were patches of ecchymosis on the mucous membrane of the stomach. The heart was flaccid, pale, and nearly empty. The large veins, and the venous system generally, were much gorged. The case of an infant of six months, which was the subject of a trial at the Aberdeen Autumn Circuit, 1853, was communicated to me by Dr. Ogston. The child died in a few hours from a dose of sixty drops of the wine of opium. The only marked appearance in the body was congestion of the brain. Although the dose was large, and death ensued, there was no decided indication of the presence of opium in the stomach. From the official report of a number of fatal cases of opium-poisoning in India, the appearances may be summarized as follows: brain turgid, lungs congested, the heart distended with liquid blood, liver and spleen engorged, mucous membrane of the stomach either natural or slightly and uniformly injected. (*Indian Report*, 1869, p. 146.)

From this account of the appearances, it will be seen that there is nothing but a fulness of the vessels of the brain, and greater or less congestion of the lungs, which can be looked upon as indicative of poisoning with opium; and even these are not always present. This condition of the brain or lungs, however, if it exist, can furnish no evidence of poisoning, when taken alone, since it is so frequently found, as a result of morbid causes, in the bodies of persons who have died from disease.

Fatal Dose.—The medicinal *dose* of opium, in *extract* or *powder*, for a healthy adult, varies from half a grain to two grains. Five grains would be a poisonous dose to most adults. The medicinal dose of the *tincture* for an adult is from *ten* to *forty minims*.

The *smallest dose of solid opium* which has been known to prove fatal to an adult, was in a case reported by Dr. Sharkey, of Jersey. (*Med. Gaz.*, vol. 37, p. 239.) A man, æt. thirty-two, swallowed two pills, containing each about one grain and a quarter of extract of opium —a quantity equivalent to *four grains* of crude opium: he was soon afterwards attacked by a convulsive fit, and died. In the case of a sailor, three drachms of the tincture proved fatal, in spite of early treatment. (Dr. Gibb, *Lancet*, July 25, 1857.) The *smallest fatal dose of the tincture* in an adult, which I have found recorded, is *two drachms*. The case is reported by Dr. Skae. (*Ed. Med and Surg. Jour.*, July, 1840.) The patient was a robust man, æt. fifty-six; he swallowed the tincture at ten in the evening, and died under the usual symptoms the following morning; the case thus lasting only twelve hours. The quantity actually swallowed, however, appears to be involved in some doubt; for it is subsequently stated that *half an ounce* of laudanum may have been taken. Opium, as meconate of morphia, was detected in the stomach. In one case which I was required to investigate, a woman died in twenty-two hours after taking *half an ounce* of tincture of opium by mistake for tincture of rhubarb. In another case, May, 1870, a lady died from a dose of two drachms of laudanum, mixed, it was supposed, with chloroform. Large doses of the tincture have frequently been taken without proving fatal. I have elsewhere recorded a case in which five ounces of laudanum were taken even without producing sleep, and the patient recovered. (*Guy's Hospital Reports*, October, 1850, p. 220.) In the *Lancet*, for March 29, 1873, p. 468, the case of a lady, æt. thirty-eight, is reported, in which it is stated that she swallowed at a dose *eight ounces* of laudanum. She was not discovered for some hours, but recovered under active treatment. Dr. Harvey, of Aberdeen, has communicated to me the case of a man of intemperate habits who swallowed *eight ounces* of laudanum. The stomach-pump was not used until six hours afterwards: he recovered under treatment. Narcotism was at no time very strongly manifested. Such cases only admit of explanation by non-absorption or idiosyncrasy.

Action of Opium on Infants.—In connection with this subject, it is important for a medical jurist to bear in mind that *infants* and young persons are liable to be killed by small doses of opium; they appear to be peculiarly susceptible of the effects of this poison. Dr. Ramisch, of Prague, met with an instance of a child, four months old, that was nearly killed by the administration of one grain of Dover's powder, containing only the tenth part of a grain of opium; the child suffered from stupor and other alarming symptoms. The following case occurred in June, 1822. Four grains of Dover's powder (containing less than half a grain of opium) were given to a child four years and a half old. It soon became comatose, and died in seven hours. Death was referred to inflammation of the throat, and the jury returned the usual unmeaning verdict of "Died by the visitation of God;" but there was no doubt, from the evidence, that death was caused by the opiate medicine. Dr. Thorn, of the Cape of Good Hope, has given me the particulars of a case of an infant only three days old, to which ten grains of Dover's powder (containing one grain of opium) were given by mistake. It suffered from the usual symptoms, but survived for the long period of forty-four hours. Dr. Kelso met with an instance in which a child, nine months old, was killed in nine hours by four drops (? minims) of laudanum, equal to less than *one-fourth of a grain* of opium: it was much convulsed before death. A case is referred to in the *Medical Gazette*, in which two drops (? minims) of laudanum, equal to about the *eighth part of a grain* of opium, killed an infant. The following is another illustration of the fatal effects of a similar dose. A nurse gave to an infant, five days old, *two drops* (? minims) of laudanum, about three o'clock in the morning. Five hours afterwards the child was found by the medical attendant in a state of complete narcotism. It was revived by a cold bath, but a relapse came on, and it died the same evening, about eighteen hours after the poison had been given to it. On inspection, the brain and abdominal viscera were found in a perfectly healthy state, and there was no smell of opium in the stomach. (*Prov. Med. Jour.*, Oct. 28, 1846, p. 519.) The fatal dose here, as in the former case, was equal to the eighth part of a grain of opium, and to only an infinitesimal dose of morphia!

Dr. E. E. Smith has reported a case (*Lancet*, April 15, 1854), in which an infant *seven days* old, died in eighteen hours from the effects of one *minim* of the tincture, or the *fifteenth part of a grain* of opium. Coma with the usual symptoms was complete in half an hour. On inspection, the heart was found distended with black liquid blood; the lungs were collapsed, but not congested. The brain was congested, but there was no effusion either into the ventricles, or on the surface. (See also *Med. Times and Gazette*, April 15, 1854, p. 386.) The smallest fatal dose recorded (in an infant) was in a case communicated to me by Dr. Edwards, of Liverpool (November, 1857). An infant, four weeks old, died from the symptoms of poisoning by opium, in seven hours after a dose of paregoric elixir, equivalent to *one-ninetieth* of a grain of opium, had been administered to it. With a knowledge of these facts, it is not surprising that infants are occasionally destroyed by opium under circumstances in which an adult would not suffer. In December, 1860, an inquiry took place at Chester respecting the death of a child, aged six weeks, under the following circumstances. A fomentation composed of laudanum and gin was applied to the side of the mother, and the child was put to the breast shortly afterwards. The child fell into a sleep from which it did not awaken, and died, in spite of treatment, the next morning. The cause of death was left obscure owing to the imperfect manner in which the inquiry was conducted; but it is not improbable that the child drew a quantity of laudanum into its mouth, sufficient to destroy life. (*Med. Times and Gazette*, Jan. 19, 1861, p. 70.) In some instances infants have been found to manifest an astonishing power of recovery. Dr. Guy met with a case in which an infant of six months recovered after having had administered to it ten grains of Dover's powder, equal to one grain of opium (*Lancet*, June 8, 1850); and Mr. Tubbs has informed me, that in a case which occurred in January, 1852, an infant of nine months recovered under treatment from a dose of two teaspoonfuls of laudanum, given by mistake. This quantity left by evaporation four grains of an impure extract of opium. In 1860, a case was communicated to me in which an infant of between two and three months old recovered after *five grains* of opium had been given to it by mistake for rhubarb. Dr. Hays met with a case in which a child, not quite six years old, swallowed a powder containing *seven and a half grains* of opium mixed with powdered chalk. The child was not seen until fourteen hours afterwards. It was at first excited; there had been no vomiting. The narcotism was at no time very profound; it gradually subsided, and at the end of three days the child had recovered! (*Am. Jour. Med. Sci.*, April, 1859, p. 367.)

Period at which Death takes place.—It has been remarked that most cases of poisoning by opium prove fatal in from six to twelve hours. They who recover from the stupor, and survive longer than this period, generally do well; but from cases above related, it would seem that there may be a partial recovery, or a remission of the symptoms, and afterwards a relapse. The symptoms, however, generally progress steadily to a fatal termination, or the stupor suddenly disappears, vomiting ensues, and the person recovers. Several instances are recorded of this poison having destroyed life in from seven to nine hours. One has occurred within my knowledge, in which an adult died in five hours after taking the drug prescribed for him by a quack. Sir R. Christison met with a case which could not have lasted above five, and another is mentioned by him which lasted only three hours. Mr. Barwis, of Melton, communicated to me the case of an adult (November, 1863), which proved fatal in three hours and a half. Nearly two ounces of laudanum had been taken; but there was no smell of opium in the stomach when inspected thirty hours after death. Dr. Beck quotes a case which proved fatal in two hours and a half. (Beck, *Med. Jur.*, p. 873.) Mr. Procter, of York, communicated to me the case of a woman, æt. 50, who in January, 1857, swallowed an ounce of the tincture, and died from the effects in less than *two hours*. Opium was found in the stomach. The only appearance in the body was a congested state of the membranes of the brain. The most rapid case of death yet reported was that of a soldier who was accidentally poisoned, in September, 1846, in the Hospital of Val de Grâce. It appears that he swallowed by mistake about an ounce of laudanum, and it is stated that he died in convulsions in *three-quarters of an hour*. (*Journal de Médecine*, Oct. 1846, p. 475. For a similar case, see *Med. Gaz.*, vol. 45, p. 743.) It is possible that the drug may destroy life even with greater rapidity than this; but, as a medico-legal fact, we are at present entitled to state that it has destroyed life within the short period above mentioned. On the other hand, the cases are sometimes much protracted. There are several instances of death in fifteen or seventeen hours. I have known one case fatal in twenty-two hours, and among those collected by Sir R. Christison, the longest lasted twenty-four hours. (*Op. cit.*, p. 712.)

Treatment.—The first object is to remove the poison by the stomach-pump, or in the case of an infant, by a catheter, as speedily as possible. This instrument should be employed until the water used for washing out the organ has no longer the color or smell of opium. The entire absence of the drug may be better indicated by adding to the liquid a few drops of a solution of a persalt of iron. If no red color be produced, there is reason to believe that there is no meconic acid, and therefore no opium present. In thus removing the poison, we at once arrest the progress of absorption. Emetics are of no service unless the person possesses the power of swallowing. Occasional doses of sulphate of zinc may then be given to him, and in the intervals a decoction of strong coffee or tea. Cold effusion on the head, chest, and spine has been adopted with great success; in the treatment of infants the plunging of the body into a warm bath, and suddenly removing it from the water into the cold air, has been found a most effectual method of rousing them. (*Med. Gaz.*, vol. 25, p. 878.) Flagellation to the palms of the hands and soles of the feet or the back, has also been successfully employed. A common plan for rousing an adult is to cause him to keep in continual motion, by making him walk between two assistants. Above all things the tendency to fall into a state of lethargy must be prevented. If called to an adult or infant already in a lethargic condition, the application of shocks to the head and spine by an electro-magnetic apparatus will be found most effectual. It has in several instances led to recovery when the person was in an almost hopeless condition. An illustration of the effects of an overdose of this drug on a child, and of the benefit derivable from the electro-magnetic treatment, is furnished by a case reported by Mr. Colahan. (*Dub. Med. Press*, April 22, 1846, p. 244.)

When there are signs of recovery, ammonia applied on rags to the nostrils, and frictions to the chest with the compound camphor liniment, will aid in restoring the patient. Injections of oil of turpentine and starch have been used beneficially to keep the person roused. Poultices of mustard and cayenne pepper have been applied to the skin with advantage. The means above stated, variously employed, have been found eminently successful; but especially the removal of the poison by the stomach-pump. Out of numerous cases of poisoning by opium brought to Guy's Hospital, but very few have proved fatal, even when the remedial treatment was applied late.

A strong decoction of coffee has been frequently employed as a stimulant to promote recovery, and apparently with benefit. Böcker affirms that, in cases in which there is a tendency to congestion of the brain and lungs, this liquid operates injuriously, and that there is no case recorded in which recovery can be assigned, in any shape, to the use of it. (*Vergiftungen*, 1857, p. 144; *Ann. de Thérapeutique*, 1847, p. 303.) It appears to have been given on the principle that it causes wakefulness, while opium produces sleep. On the same grounds he condemns the use of brandy and camphor. From some cases which have occurred, it would appear that alcohol in any form is objectionable. Coffee with the stomach-pump, the ambulatory treatment, and cold effusion to the head and face, has proved most beneficial. Dr. Johnson found that great benefit resulted in cases of severe opium-poisoning from the hypodermic injection of from a quarter to half a grain of atropia. (*Am. Jour. Med. Sci.*, July, 1873, p. 279.)

CHAPTER LVI.

COMPOUNDS CONTAINING OPIUM—POISONING WITH POPPIES—SYRUP OF POPPIES—GODFREY'S CORDIAL—DALBY'S CARMINATIVE—PAREGORIC—DOVER'S POWDER—BLACK DROP—SEDATIVE SOLUTION—WINE OF OPIUM—OPIUM LOZENGES—CHLORODYNE—NEPENTHE.

POISONING BY POPPIES.

THE heads of the white poppy, grown in this country, contain meconate of morphia. They yield an inspissated extract, called English opium, which, according to the late Mr. Hennell, contains five per cent. of morphia. The white poppy-heads, therefore, yield to water, in the form of decoction, a poisonous salt capable of acting deleteriously on infants or children. Among several cases illustrating the noxious effects of poppy-heads to infants, the following may be mentioned. Two children in a state of narcotism were brought to a medical man. On inquiry it was found that, with a view of procuring sleep, the mother had boiled a poppy-head in water, and had given to each child one or two teaspoonfuls of the decoction. In spite of treatment, one child died seven or eight hours after it was first seen. The decoction had the usual opiate smell. (*British Med. Journal,* Oct. 31, 1857, p. 909.) In July, 1863, a child died at Bilston, in consequence of its mother having administered to it, two spoonfuls of water in which a poppy-head had been boiled. (*Med. Times and Gaz.,* July 18, 1863, p. 75.)

Many cases of poisoning have occurred from the injudicious use of *Syrup of poppies,* which is nothing more than a sweetened decoction of the poppy-heads. This syrup, in its ordinary state, is said to contain *one grain* of extract (opium) *to one ounce* (Thomson). The common dose of it for an infant three or four months old, is half a drachm; for adults, two to four drachms (Pereira). There is reason to believe the compound sold by some druggists for syrup of poppies, as a soothing or cordial medicine for children, is nothing more than a mixture of tincture or infusion of opium with simple syrup; it is therefore of variable strength. This may account for what appears to many persons inexplicable, namely, that an infant may be destroyed by a medicinal dose. (*Med. Gaz.,* May, 1831, p. 253.) In January, 1841, a child six months old is said to have died from the effects of less than half a teaspoonful of syrup of poppies bought at a retail druggist's. The symptoms of narcotic poisoning were fully developed in three-quarters of an hour. The syrup in this case probably contained tincture of opium. Seven children are reported to have lost their lives by this syrup in 1837–8. In one of these cases, a teaspoonful and a half was given. Stupor came on in half an hour, and the child died the following day. The late Dr. Pereira states that he has known a teaspoonful to prove fatal to a healthy child. (*Mat. Med.,* vol. 2, part 2, p. 644.)

The late Dr. Burke Ryan communicated to me the particulars of a case which proved fatal from a similar dose in December, 1854. A drachm of the syrup was given by a mother to her child, aged fifteen months. In four hours the child was found in a deep sleep, with stertorous breathing—the face cold and pale—the body motionless, and in this state it died, eight hours after taking the syrup. I found by examination that the syrup was such as is usually sold in druggists' shops. The principal appearances in the body were congestion of the membranes and sinuses, but not the substance of the brain. There was a reddish-colored fluid in the ventricles. The heart was firm, and the right cavities were filled with half-coagulated black blood. The stomach and intestines were healthy. (*Lancet,* Jan. 20, 1855, p. 68.)

I have notes of two cases which occurred in 1871, in one of which a child eighteen weeks old died in twenty-six hours from the effects of a teaspoonful, and the other in which an infant five weeks old was killed by three parts of a teaspoonful of this syrup. It is in all cases a most uncertain preparation as to strength.

Mrs. Winslow's *Soothing Syrup,* called also "Quietness," appears to be a compound resembling syrup of poppies. Its effects are those of a narcotic. Two doses of this caused the death of a child aged fifteen months, with the usual symptoms of narcotic poisoning (*Pharm. Jour.,* June, 1872, p. 618.) An analysis of this syrup showed that one ounce of it contained nearly one grain of morphia with other opium alkaloids. (*Pharm. Jour.*, 1872, p. 975.) It is not surprising that it should prove fatal to infants in small doses. Another fatal case from the use of this syrup is reported in the *British Medical Journal* for Sept. 27, 1873, p. 380. The reporter suggests that there should be some power to stop the sale of a poison which is sold under the false pretence that it is perfectly safe!

The following are cases of poisoning by the *Decoction of poppies*. A woman boiled two poppy-heads in a quarter of a pint of milk, and gave only two small spoonfuls of this decoction to her child. In an hour, the child fell into a lethargic sleep, the respiration was stertorous, and in ten hours it died. On inspection, the brain and its membranes were found congested. In a second a case, a maidservant, in order to quiet a child, gave to it two teaspoonfuls of a decoction made by boiling one poppy-head in a small quantity of water. The child was found dead in the morning. The brain and its membranes were much congested, and the ventricles contained bloody serum. The seeds of the poppy were found in the stomach.

In the following case, the symptoms were of a serious character, but the child recovered. A woman gave to her child several teaspoonfuls of a strong decoction of poppy-heads. In a quarter of an hour it fell into a deep sleep, from which it could not be roused. Medical assistance was not called for thirty-six hours, and then the child was apparently dying. The eyes were sunk, the lids half open and surrounded by a livid circle, the pupils *dilated* and insensible, the face pale, with a slight bluish tint, the extremities almost paralyzed, respiration hurried, the pulse frequent, small, and trembling, the forehead covered with a cold sweat, and the lower jaw depending. No urine had been voided, and there had been no evacuation from the bowels since the first occurrence of the symptoms. It was then too late to think of removing the poison from the stomach. Coffee and other stimulants were used, under which the child recovered. (*Ann. d'Hyg.*, 1845, vol. 1, p. 212; also *Med. Gaz.*, vol. 36, p. 305.)

It may be observed that the poisonous salt of morphia is generally considered to exist in the *capsule* of the poppy, and not in the seeds; but Sobernheim mentions one or two cases of poisoning by the seeds of the plant. (*Tox.*, p. 500. For other cases, see Henke, *Zeitschrift der S. A.*, 1844, vol. 1, p. 302.) *Extract of poppies* acts like the decoction, but it is more powerful. The dose of this medicinal preparation for adults is from two to five grains.

GODFREY'S CORDIAL.

This is chiefly a mixture of infusion of sassafras, treacle, and tincture of opium. The quantity of tincture of opium, according to the late Dr. Paris, is about one drachm to six ounces of the mixture, or *half a grain of opium to one ounce*, but it is probable that, like the so-called syrup of poppies, its strength is subject to great variation. A case is reported, in which half a teaspoonful, = $\frac{1}{32}$d part of a grain of opium, was alleged to have caused the death of an infant. In 1837–38, twelve children were killed by this mixture alone, and in five years (1863–67) fifty-six deaths were recorded to have taken place from this compound. The explanation of this great mortality is, that the medicine is usually given in large doses by ignorant persons.

DALBY'S CARMINATIVE.

This is a compound of several essential oils and aromatic tinctures in peppermint-water, with carbonate of magnesia and tincture of opium. According to the late Dr. Paris, there are *five minims* of the tincture, or from one-third of a grain of opium, in rather more than *two ounces* of this mixture, or the *one-sixth of a grain* in an ounce. The formula commonly given is, carbonate of magnesia two scruples, oil of peppermint one minim, of nutmegs two minims, of aniseed three minims, tincture of opium five minims, spirit of pennyroyal and tincture of assafœtida of each fifteen minims, tincture of castor and compound tincture of cardamoms of each thirty minims, and of peppermint-water two ounces. According to this formula, tincture of opium forms the 211th part by measure, or on the pharmaceutical strength, one teaspoonful would contain the 64th part of a grain of opium. Like most of these quack preparations, it no doubt varies in strength. An infant is reported to have been destroyed by *forty drops* of this nostrum—a quantity, according to the strength assigned, equivalent to more than *two minims* of the tincture, or from one-sixth to one-tenth part of a grain of opium. Accidents frequently occur from its use, partly owing to ignorance, and partly to gross carelessness on the part of mothers and nurses.

The quack medicine, known under the name of *Locock's pulmonic wafers*, contains opium. A boy, æt. 4, suffered from all the usual symptoms of poisoning by opium as a result of eating a quantity of these wafers or lozenges. (*Lancet*, Oct. 27, 1860, p. 420.)

PAREGORIC ELIXIR—CAMPHORATED TINCTURE OF OPIUM.

This is a medicinal compound of alcohol, opium, benzoic acid, oil of aniseed, and camphor. Opium is the active ingredient, and of this, the tincture contains *one grain* in every *half ounce*. It is sold to the public at the rate of fourpence per ounce. Fatal cases of poisoning by paregoric are not very frequent: the following case was referred to me some years since. A child between five and six years old, had had some cough medicine prescribed for it at a druggist's. The medicine consisted, as nearly as could be ascertained from a portion left in a bottle, of paregoric, having about from one-fourth to one-half the strength of the pharmacopœial tincture. The child took two-thirds of this mixture, given in divided doses, at somewhat irregular intervals, and died in about thirty-six hours. The quantity of opium in the portion of the mixture taken was, judging by comparison with the usual strength of the tincture, from three-fourths of a grain to one grain and a quarter. The child was drowsy after each dose, and slept on one occasion for several hours together. It was occasionally roused, and appeared sensible; but again relapsed into drowsiness on taking the medicine. A few hours before its death, it was found comatose, with stertorous breathing and strongly contracted pupils. On inspection the whole of the organs were healthy, with the exception of the parietes of the ventricles of the heart, which were somewhat thickened; there was no congestion of the vessels of the brain, nor effusion in the ventricles. The liquid contents of the stomach yielded no trace of opium or an opiate. There was, however, no doubt that the death of this child had been caused by an opiate. This was proved, 1, by the nature of the medicine taken; 2, the nature of the symptoms, which were aggravated after each dose; 3, the confirmed coma and stertor; and, lastly, the absence of any other cause to account for the symptoms and rapid death under the circumstances. (*G. H. Reports*, April, 1844.) It was a question here how far a small quantity of opium in divided doses was likely to prove fatal to a child of this age. The answer was given to the effect that, although each dose might be individually harmless, the frequent repetition of the medicine, when the child had scarcely recovered from the effects of a previous severe dose, might operate fatally. (See paper by Dr. Beck, *Med. Gaz.*, vol. 33, p. 771.)

In another case a child, aged seven months, was killed by a teaspoonful given in two doses at an interval of a day, *i. e.*, by a quantity equal to one-quarter of a grain of opium. (*Pharmaceutical Jour.*, April, 1845, p. 464.)

In another, an infant of five weeks recovered from a similar dose, although no treatment was resorted to for nine hours. (*Med. Times and Gaz.*, Aug. 6, 1859, p. 145.) Dr. Edwards, of Liverpool, informed me of a case in which an infant had been killed by a dose equivalent to not more than the *ninetieth* part of a grain of opium. Dr. Beck has recorded an instance in which a child was narcotized by about twenty drops of this tincture, = $\frac{1}{12}$th grain of opium, or at a maximum, $\frac{1}{120}$th grain of morphia. (*Medical Gazette*, March, 1844, vol. 33, p. 767.) Aged persons may also die from the effects of small doses. At a recent inquest the deceased, a woman, æt. 77, laboring under chronic cough, was proved to have died from the effects of a dose of two drachms of this liquid, equal to about half a grain of opium.

The *ammoniated tincture of opium* is compounded of opium, saffron, benzoic acid, oil of anise, strong solution of ammonia, and rectified spirit. It contains one grain of opium in ninety-six minims, and the medicinal dose is from half a drachm to one drachm.

DOVER'S POWDER (COMPOUND POWDER OF IPECACUANHA).

This is a preparation of opium, the effects of which on children have been already described (p. 535). The proportion of opium is one-tenth part, or *one grain* in every *ten grains* of the powder. A child has been killed by four grains, therefore by a quantity containing about two-fifths of a grain of opium.

Symptoms and Appearances.—The following case of poisoning by Dover's powder occurred to Mr. Griffiths. (*Medical Gazette*, March, 1844.) About ten grains of the powder (equivalent to *one grain* of opium) were given by mistake to an infant seven weeks old, and it died in twenty-four hours. On an inspection of the body, the countenance was placid, and the fingers of both hands were firmly contracted. In the abdomen, the spleen, kidneys, and intestines were found in a healthy condition; the liver was gorged with blood; the stomach contained a small quantity of a colorless viscid matter, in which neither morphia nor meconic acid could be detected. The inner coat was reddened; and at the great curvature, as well as in other parts, the bloodvessels were highly injected in patches. The lungs were gorged with blood; the upper lobes being infiltrated with a greenish serum. The pericardium was reddened, and contained about a drachm of fluid. The right auricle was empty; the left ventricle contained some thin fluid blood, and a small coagulum. The sinuses of the dura mater were filled with dark coagula; the surface of the brain appeared covered with a complete network of vessels, distended with light-colored blood. On the surface of each posterior lobe there was a slight effusion of blood. The brain was soft, and the difference of color between the gray and white matter barely discernible. The vessels in the substance of the brain were gorged with blood, presenting, on section, a thickly studded appearance; the spots were of a deep dull red, and in many places coalescing. There was a small quantity of fluid in each lateral ventricle, and on the floor of each the bloodvessels were largely distended. There was an effusion of serum on the surface as well as at the base of the brain, to the amount of half an ounce. The contents of the stomach were carefully analyzed, but neither morphia nor meconic acid could be found.

Dover's powder enters into the composition of some *teething powders*, and, unless prescribed with great caution, it may cause fatal accidents among children. In the *Pharmaceutical Journal*, Nov. 7, 1874, p. 375, four cases are reported in which children died, narcotized by these powders. The symptoms were of the usual kind, rapid insensibility, stupor, and death. On inspection, there was congestion of the brain and its vessels. According to Mr. Wright, who saw one of the children, the druggist supplied by mistake the hydrochlorate of morphia. (*Brit. Med. Jour.*, Dec. 26, 1874, p. 835.) Dr. Brown, of Lahore, relates the case of a child at fourteen months, who took by mistake six grains of Dover's powder (equivalent to six-tenths of a grain of opium) at six o'clock P.M. In a quarter of an hour he felt drowsy, and fell asleep; at two o'clock A.M., eight hours after taking the poison, he had severe convulsions; his pupils were dilated, and his pulse was slow and irregular. He remained insensible, and died at three A.M., nine hours after taking the powder. (*On Poisons in the Punjab*, 1863, p. 71.) On the other hand, Mr. Ewens met with a case in which an infant of nine months recovered from a dose of five grains. (*Med. Times and Gaz.*, May 19, 1860.) Dr. Guy has reported another, in which an infant of six months recovered, under active treatment, from a dose of ten grains. (*Lancet*, June 8, 1850.) I am indebted for a still more remarkable instance of recovery to Mr. R. Read, of Dublin. Fifteen grains of Dover's powder were given to an infant under five months of age. The mistake was discovered immediately, and by active treatment the child recovered. Assuming that the powder contained its proportions of opium, these cases of recovery in infants must be regarded as quite exceptional.

BLACK DROP.

This is a preparation of opium, in which the meconate of morphia is combined with acetic acid. In the black drop, according to Pereira, verjuice, the juice of the wild crab, is employed as a menstruum instead of vinegar. The black drop is considered by some to have from three to four times, but according to Dr. Neligan twice, the strength of the tincture of opium. A formula for this preparation will be found in Dr. Neligan's work, *On Medicines*, etc., p. 275. According to this, it is a compound of half a pound of opium to three pints of the expressed juice of the wild crab, with nutmegs, saffron, and sugar. It resembles the *acetum opii*, and has more than twice the strength of laudanum.

BATTLEY'S SEDATIVE SOLUTION.

This, according to Pereira, is an aqueous solution of opium, with a little spirit and less meconic acid than in the common tincture. (*Op. cit.*, vol. 2, pt. 2, p. 646.) Others represent it as an aqueous extract of opium dissolved in rectified spirit. It is considered to have three times the strength of tincture of opium, but there is so great a difference of opinion on this point, that Dr. Neligan represents it as being of only about the same strength as laudanum. (*Medicines, etc.*, p. 276.) He states that it is composed of three ounces of extract of opium, six drachms of spirit, and as much distilled water as will make up two pints. It may be regarded as an aqueous solution of meconite of morphia (without the resin), and with just sufficient spirit to preserve it. It appears to be an uncertain preparation. Mr. Streeter stated, at the Westminster Medical Society, Dec. 1838, that he had known one drachm and a half of it to prove fatal to a lunatic; and twenty minims of the solution destroyed the life of an old woman. A medical gentleman, lying dangerously ill from an attack of dysentery, took, by mistake, about seven drachms of Battley's solution. Within five minutes, salt and water, with mustard, were administered, and twenty-four grains of sulphate of zinc. Vomiting ensued; the emetic was repeated, and with the same effect; the fluid evacuated at the second vomiting having the usual smell of opium. Half a drachm of ipecacuanha was afterwards given to complete the emptying of the stomach. Notwithstanding this repeated vomiting, symptoms of narcotism presented themselves speedily, with contraction of the pupils, and very great drowsiness—rendering it necessary to remove the patient from bed in his very debilitated state, and to keep him constantly moving, until 9 P.M. (seventeen hours), when vomiting came on spontaneously; he was then put to bed, and allowed to sleep. The original disease afterwards resumed its course (complicated by an attack of gastritis), and at length terminated favorably; but the patient had no recollection of what had occurred for twenty-four hours after the administration of the emetics; and it appeared to his medical attendants that an excited state of the mind remained for some days afterwards. (*Prov. Jour.*, Jan. 28th, 1846, p. 42.) The death of Dr. Baddeley, of Chelmsford, from a medicinal dose of this solution, furnishes an additional proof of the dangerous uncertainty of the strength of this solution.

WINE OF OPIUM (VINUM OPII).

This is a pharmacopœial compound of extract of opium, cloves, cinnamon, and sherry wine. It was formerly known as Sydenham's laudanum, or *Laudanum liquidum Sydenhami.* For internal use, the dose is from ten to forty minims. It contains twenty-two grains of extract of opium nearly in one ounce; its effects in poisonous doses are similar to those produced by the tincture. It is rarely used as a poison. (See *Laudanum*, p. 528.)

OPIUM LOZENGES.

Some of the cough lozenges sold to the public contain opium. Mr. Garlick communicated to the *Lancet* a case in which he was called to a man who had occupied himself, during an afternoon, in sucking one ounce and a half of these lozenges. After a time he was observed to become drowsy. His countenance was pale; there was great somnolency; and it was with difficulty that any reply to a question could be obtained. The pupils were strongly contracted; the breathing heavy and oppressed, occasionally stertorous; and the pulse small and feeble. With some difficulty, and after active treatment, the man recovered; but for a period of twenty-four hours he experienced general numbness. The vendor of the lozenges knew nothing about the quantity of opium contained in them! (*Lancet*, p. 137, Jan. 30, 1847.)

CHLORODYNE.

A fatal case from an overdose of this medicine occurred at Oxford in 1871. A lady, æt. 23, had been accustomed to take this liquid for the relief of pain, in doses of as much as sixty drops. She was found dead in bed, and the cause of death was referred by her medical attendant to her having taken two doses without letting a sufficient interval elapse between them. (*Lancet*, 1871, vol. 2, p. 697.) From 1863 to 1867, four deaths were caused by this compound. According to Mr. E. Smith, chlorodyne is thus constituted: Chloroform four drachms, muriate of morphia twenty grains, rectified ether two drachms, oil of peppermint eight minims, prussic acid six drachms, mixture of gum acacia one ounce, treacle four ounces. (*Lancet*, 1870, vol. 1, p. 72.) There is reason to believe that this compound is not uniform in composition. According to another formula, the tinctures of lobelia and capsicum, with extract of liquorice, are introduced. I found that one sample, on standing, separated into two liquids, one light and of a pale straw color, and the other heavy and of a brown color and syrupy consistency. On evaporation, it left half of its weight of solid residue as saccharine matter. Another sample remained in a thick syrupy state. A fluid drachm of the first sample left as a residue twenty-seven grains of a brown saccharine extract. Prussic acid was detected in it by the reaction of the vapor on nitrate of silver. Crystals of cyanide of silver are obtained after some time. Morphia may be detected in it by shaking a portion with a mixture of sulphide of carbon and iodic acid. The sulphide acquires a pink color, owing to the separation of iodine by the morphia. The other substances may be detected by their appropriate tests.

NEPENTHE.

In a case tried at the Chester Assizes, the nature of the compound actually sold under the above name was a subject of inquiry. It is a pale sherry-colored liquid, of a spirituous odor, with the smell of opium, and an acid reaction. It contains 3.4 per cent. of solid matter, consisting chiefly of a purified extract of opium, citrate of morphia, and a small quantity of grape-sugar. It also contains a trace of tannic acid, derived from sherry, which is a large constituent. This causes a darkening of the liquid when a persalt of iron is added to it for the detection of meconic acid. This disappears and the red color of meconate of iron is brought out when a small quantity of diluted sulphuric acid is added to it. Morphia is readily detected in it by a mixture of iodic acid and sulphide of carbon. The sulphide acquires a pink-red color. Although the formula for its preparation has not been published, Nepenthe may be regarded as a solution of opium and citrate of morphia, in nearly equal parts of sherry and rectified spirit, diluted with half its volume of water. The morphia is in larger proportion than the opium. It constitutes $\frac{1}{74}$th part of the compound, while the opium forms the $\frac{1}{98}$th. As a medicine and a poison it acts like laudanum, and is considered to have about the same medicinal strength. The morphia is in perfect solution, and therefore in a state fit for rapid absorption. A very small quantity has sufficed to destroy the life of an infant. An infant fourteen days old died under the following circumstances. Some dill-water was procured at a druggist's, and it was put into a bottle which had contained nepenthe. A teaspoonful was given to the infant. It soon fell asleep, and died in a short time under all the symptoms of narcotic poisoning. The dill-water was slightly colored by the small quantity of opiate which it had dissolved. (*Pharm. Jour.*, 1872, p. 779.)

CHAPTER LVII.

POISONING WITH MORPHIA AND ITS SALTS—SYMPTOMS AND APPEARANCES—FATAL DOSE—TREATMENT—NARCOTINA—CODEIA—THEIR CHEMICAL AND PHYSIOLOGICAL PROPERTIES.

MORPHIA AND ITS SALTS.

MORPHIA is the poisonous alkaloid of opium, of which it forms from five to ten per cent. The two principal salts of this alkaloid are the HYDROCHLORATE and the ACETATE. The pharmacopœial solutions of these salts of morphia contain one grain in two fluid drachms of each. In five years (1863–7) there were thirty-two deaths from morphia in England and Wales.

Symptoms.—The symptoms generally *commence* in from *five* to *twenty minutes* after the dose of poison (in solution) has been swallowed; and they closely resemble those observed in poisoning with opium. As a summary, it may be stated that they consist of dimness of sight, weakness and relaxation of the muscular system, the face and hands congested and of a livid or bluish color, the skin cool, a strong tendency to sleep, stupor, loss of consciousness, coma, stertorous respiration, and, more commonly than in poisoning by opium, there are convulsions. According to Orfila, in nineteen-twentieths of all cases the pupils will be found strongly contracted, a statement which I believe to be correct; the few exceptional cases were those in which the dose was excessive, and the symptoms were unusually violent. The state of the pupils gave rise to a great difference of opinion among the medical witnesses on the trial of Dr. Castaing. (*On Poisons*, 2d ed., p. 619.) The condition of the pulse varies greatly. It has been found small and feeble, sometimes full and slow. In some cases there is great irritability, as well as itching of the skin, and irritability of the bladder with difficulty of passing urine. Vomiting and purging have been met with in those instances in which the dose was large.

It has been stated that morphia in large doses does not operate as a narcotic, but as a stimulant to the nervous centres, causing violent convulsions. In some instances the convulsions are said to have assumed a tetanic character, resembling those caused by strychnia. This is said to have been noticed where the alkaloid or its salts had been used hypodermically. The statement appears to be based more upon theory than fact. I know of no cases to support the theory, but many adverse to it. Such theoretical views become of serious import to medical evidence, when it is pretended that the tetanic symptoms of strychnia are not to be distinguished from those caused by large doses of morphia! They just serve the purpose of unsettling everything and settling nothing. One medical authority has announced that all the symptoms assigned to poisoning by strychnia in Cook's case (*Reg.* v.

Palmer) might be explained by supposing that he had taken three grains of morphia! In this case there were no symptoms of any kind for three-quarters of an hour after deceased had taken two pills which were given to him by the prisoner. Tetanic symptoms of a violent kind with opisthotonos then came on suddenly; there was no loss of consciousness, and death occurred in twenty minutes. If this was poisoning by morphia, then medical experience and observation go for nothing in reference to poisoning by strychnia. Such a theory carries with it its own refutation.

Poisoning by morphia may take place as the result of external application. I am indebted to a friend for a remarkable illustration of its fatal effects by absorption. In September, 1867, a woman, suffering from cancer of the breast in a state of ulceration, applied to a druggist at Bungay for some medicine to relieve pain. He applied at once *thirty grains* of morphia, covering with it the surface of the ulcer. The woman soon after became insensible. When seen by a medical man she was quite unconscious—the pupils were contracted, the skin very cold, the pulse full and compressible. The woman was then in a hopeless state, and she died in ten hours after the application of the morphia to the breast. The druggist, when examined at the inquest, said that in his judgment the application was right and proper, and in spite of medical evidence that the symptoms and death were referable to morphia by absorption, the jury returned a verdict of death from natural causes.

Appearances after Death.—The only post-mortem appearance which can be referred to the action of morphia is fulness of the cerebral vessels, with occasionally serous effusion and bloody points on a section of the brain substance. This poison has no local irritant action, and it therefore leaves no marks of its operation in the stomach and bowels. An account of the appearances produced by an overdose of sulphate of morphia has been published by Orfila in a report of the case of Dr. Ellenberger (*Ann. d'Hyg.*, 1852, vol. 2, p. 359). The case presents some curious features. The deceased imagined that he had discovered a certain antidote for morphia and its salts, and proposed, while Orfila was at Prague, in October, 1851, to swallow the poison and the antidote in his presence. Orfila consented to witness the experiment. A powder was produced, which was found to have a bitter taste, and to possess some of the chemical properties of morphia, evidently mixed, however, with some other substance. The doctor swallowed about twenty-three grains of this powder, and immediately afterwards his so-called antidote, which was a fine white powder, having a sweetish taste. He did not suffer from any symptoms of poisoning. Orfila, with a keen eye to the practical use of antidotes, inquired whether he had ever allowed a certain interval to pass before taking the remedy. Dr. Ellenberger said that the results were the same. Six months after this experiment, Dr. Ellenberger died from a dose of about ten grains of sulphate of morphia. He had taken his antidote, but not until a considerable interval had elapsed! A minute inspection of the body was made, and the principal appearance was a well-marked congestion of the brain and its membranes. There were traces of sulphate of morphia in the stomach.

The so-called antidote was examined, and found to consist of a mixture of magnesia and carbonate of magnesia! Dr. Ebertz, of Weilberg, lately met with a case in which an overdose of the hydrochlorate, supplied by mistake for quinine, destroyed the life of a lady in from forty to fifty minutes. Symptoms of narcotism appeared in a quarter of an hour. A very full account of the appearances and analysis will be found in Eulenberg's *Vierteljahrs.*, 1873, vol. 1, p. 281.

Fatal Dose—Period of Death.—Five cases are recorded in which a dose of *one grain* of hydrochlorate of morphia has proved fatal to adults; in one it was taken in solution; in a second in a pill; in a third in a powder; and in a fourth it was administered by hypodermic injection into the tissue under the skin of the forearm. The first of the cases occurred to Dr. Paterson in December, 1845. (*Ed. Monthly Journal*, Sept. 1845, p. 195.) The morphia was taken in divided doses, in six hours. The symptoms and appearances were of the usual character, and insensibility came on rapidly. The patient died in about seven hours. The second case occurred at St. Mary's Hospital, May, 1861; a man, æt. 45, died in thirteen hours from a dose of one grain of hydrochlorate of morphia, prescribed in a pill by one of the physicians. The symptoms came on in about three hours, and were of a well-marked character. No morphia was detected in the stomach or other organs, and its operation as a poison was ascribed, without any apparent ground, to disease of the kidneys. In the third case (March, 1863) I was consulted by Mr. Charsley, Coroner for Bucks. A healthy man, æt. 52, died in about ten hours from the effects of *one grain*. Three hours after taking the powder, he lost his senses of smelling and hearing, and passed rapidly into a comatose condition, from which he did not recover (case of Cordery, Burnham, March, 1863). The practitioner who prescribed the morphia alleged that he had given only half a grain, but the facts of the case were adverse to this statement. On analysis I could detect no morphia in the stomach. The fourth case occurred at the Middlesex Hospital, in May, 1863. I am indebted for the particulars to Mr. De Morgan, under whose care the patient was placed. One-third of a grain of morphia was injected at night under the skin; in two hours the injection of a similar quantity was repeated. On the next morning another third of a grain was injected. The man slept quietly for two hours. He then took some dinner, and talked in his usual way; but in another hour he became almost suddenly insensible, and in two hours he died, the narcotic symptoms being most powerfully developed. In a fifth case, reported in the *Lancet*, 1872, vol. 2, p. 24, a lady died in less than twelve hours from taking a pill containing one grain of morphia. Galvanism and other methods were used for restoration, but without success. The druggist sent six pills with a grain in each, instead of dividing the grain into six pills! (*Lancet*, 1872, vol. 2, p. 24.) The late Dr. Anstie met with a case in which three grains of morphia given as an injection per rectum caused death in sixteen hours.

The hydrochlorate is thus proved to be a powerful poison in a small dose: it may operate either suddenly or slowly, and destroy life rapidly. An infant has died from a dose of one-twelfth part of a grain. (*Chem. News*, Aug. 22, 1863, p. 98.) Sir R. Christison considers that one grain of the hydrochlorate is fully equal in power to six grains of the best Turkey opium. There is no reason to suppose that the acetate is less potent; but there are some remarkable instances of recovery where such a result could scarcely have been anticipated. In the *Lancet* for 1863, vol. 1, p. 8, is reported the case of a child about two years of age, who recovered from a dose of one grain of the acetate. The medicinal dose for an adult of either of these salts of morphia is from one-eighth of a grain to one-half grain. A case in which *one grain* of the acetate, dispensed by mistake in a pill, destroyed the life of a lady, is reported. (*Pharm. Jour.*, July, 1872, p. 16.) Narcotic symptoms came on in about half an hour, and she died in nine hours.

The small quantity of morphia required to destroy life is not sufficiently known to members of the profession. In *Reg.* v. *Macleod*, Cumberland Lent Assizes, 1874, it was proved that the deceased, the wife of the accused, had died from the effects of hydrochlorate of morphia. The accused was surgeon-major in the Madras army. He had prescribed and administered the hydrochlorate of morphia to his wife, and the charge against him was that he had acted with criminal negligence, and had thus led to her death. It appeared that in consequence of the deceased having passed some restless nights, the prisoner procured from a druggist twenty grains of muriate of morphia. He gave her one grain at four o'clock P.M., and as this appeared to have no effect, he gave her several unknown doses at half-hour intervals, using at least one-half of the morphia which he had purchased. Deceased was thrown into profound coma, from which nothing could rouse her, and she died at ten P.M. On inspection the chief appearances were congestion of the brain and fluidity of the blood. The organs were healthy, and there was nothing to account for death but the morphia. The jury acquitted the prisoner of the charge of manslaughter, finding that there had been no culpable negligence.

This case shows the importance to members of the medical profession of a knowledge of the difference between a medicinal and poisonous dose of morphia, and the necessity for allowing proper intervals to pass between the doses.

Homœopathic practitioners have the repute of employing doses so minute that the most delicate process of analysis fails to show by chemical tests the presence of any of the substance. This, however, is not always true. In July, 1847, the late Sir John Forbes consulted me in the case of an aged lady to whom he was called, and whom he found in a state of stupor, with contracted pupils, and other symptoms of narcotic poisoning. The facts were simply these. A homœopath had prescribed for her some powders, each of them numbered, with explicit directions as to the order in which they were to be taken. The lady had taken two, and it was after the alarming narcotic effects produced by the second, that Sir J. Forbes was called in. He took possession of three of the powders and brought them to me. They were small white powders unequal in weight: one weighed 3.4 grains, and consisted of calomel and morphia, the morphia, as separated by alcohol, being in the proportion of *one grain*. The next powder, following the numbers, weighed 1.5 grain; it contained neither calomel nor morphia, but consisted of sugar of milk. The third powder weighed 2 grains; it consisted of calomel and morphia, the latter being in the proportion of half a grain, an allopathic medicinal dose. The cause of this lady's symptoms were thus satisfactorily explained. The powder she had taken contained morphia in an almost poisonous dose, and it had produced the usual effects. Supposing there had been only one left, *i. e.*, the powder containing sugar of milk, a perfectly harmless substance, a coroner's jury summoned to inquire into the cause of death would have been quite satisfied from the analysis of the powder that all were innocent, and their verdict would have been "Death from Apoplexy."

Treatment.—The treatment in a case of poisoning with morphia is the same as that required in poisoning with opium (*ante*, p. 537).

There are other alkaloids besides morphia which have been extracted from opium, but as poisons, they are scarcely known. Two only require a brief notice in this place, namely, Narcotina and Codeia. It will be convenient to dispose of them before describing the analysis of morphia and its salts.

NARCOTINA.

The results of experiments with this substance on animals are very conflicting. In the human subject it has been observed to produce headache; but when these effects have followed, it has been probably mixed with morphia. I have frequently found this mixture in specimens; and Sir R. Christison states that he has met with narcotina in morphia, a circumstance which may tend to explain the variable effects of morphia in large doses.

Analysis.—This alkaloid is not commonly sought for in medico-legal investigations, but it is proper to state the chemical differences which exist between it and morphia. Its crystals are rhombic prisms and have a bright pearly lustrè. It is not very soluble in water, but it is dissolved by boiling alcohol and ether. Its solutions have no alkaline reaction, and are very bitter. Unlike morphia, it is not very soluble in potash, ammonia, soda, or lime-water, all of which precipitate it from its solutions. It is not readily dissolved by diluted acetic acid even on boiling. When nitric acid is poured on the crystals, they acquire a *yellow*, not an orange-red color, like morphia. Sulphuric acid gives to narcotina a bright sulphur-yellow color; to morphia a pinkish-brown tint. If to the mixture of acid and alkaloid, a crystal of bichromate of potash be added, green oxide of chromium is set free in both cases, but very slowly in the case of narcotina. Sulphomolybdic acid produces with narcotina a pale-red color. If to the mixture of sulphuric acid and the alkaloid, a grain of nitre is added, a deep blood-red color is slowly brought out with narcotina, but not with morphia. Narcotina also differs from morphia in not decomposing iodic acid, or setting iodine free. When heated on platinum, narcotina, like morphia, melts and burns, and, if not overheated, sets into a crystalline mass on cooling.

CODEIA.

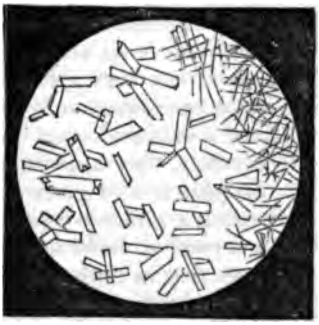
Codeia is found to exert a poisonous action on animals; it has been used in France as a narcotic. It is considered to have only one-half of the strength of morphia, with which it is in general mixed. The common hydrochlorate of morphia of the shops is, according to Pereira, a compound of morphia and codeia. M. Kunckel found that, when combined with acids, it lost much of its activity.

The only case of poisoning with this alkaloid which I have met with is the following, which occurred in the practice of Dr. Myrtle, of Harrogate. The patient was laboring under diabetes, and he prescribed for him *four grains* of *codeia* and one-twentieth of a grain of sulphate of strychnine, in the form of a pill—one to be taken night and morning. The druggist thought the dose excessive, but on the authority of Dr. Gregory, Dr. Myrtle desired the pills to be made as ordered.

In about half an hour after swallowing one, the patient felt a general glow all over him. He walked about for two hours. He then began to write, but found that he could not see the letters distinctly. Suddenly his sight failed him, he felt the room going round with him, and he then rushed into the open air. For hours after he had taken the pill, he was standing holding on by the back of a chair; his face was pale; he had a terrified look; was perspiring copiously; the pupils were slightly contracted; the pulse scarcely perceptible; skin cold and clammy; his voice reduced to a whisper. He complained of feeling very sick, but was not sleepy. He remained in this state the whole of the day, and if he attempted to move he began to retch violently. He had no sleep at night, and was slightly delirious. He continued ill the following day, but in thirty-six hours all the disagreeable symptoms had disappeared. (*Brit. Med. Jour.*, April, 1874, p. 478.)

Analysis.—This alkaloid, which is not often seen so well crystallized as morphia and narcotina, is known from both by its ready solubility in water, and by its forming a strongly alkaline solution. It crystallizes in quadrangular prisms (Fig. 53). One hundred parts of water at 60° dissolve one part and a quarter; at 212° nearly six parts. It is soluble in alcohol, and combines with acids. It differs from morphia in not decomposing iodic acid, and in not giving any red color with nitric acid, either as a solid or when dissolved in acids. It merely acquires a light orange-yellow color. It differs from narcotina in not being turned yellow, but of a light pinkish-brown color, by sulphuric acid; but it resembles both morphia and narcotina in producing green oxide of chromium when a crystal of bichromate of potash is added to the acid mixture. Sulphomolybdic acid produces with it a greenish-blue color. Heated on platinum it melts, forming a globule of colorless liquid; this soon darkens, and gives off a vapor which burns with a yellow smoky flame. (*Brit. Med. Jour.*, April 11, 1874, p. 471.)

Fig. 53.



Crystals of codeia, magnified 70 diameters.

CHAPTER LVIII.

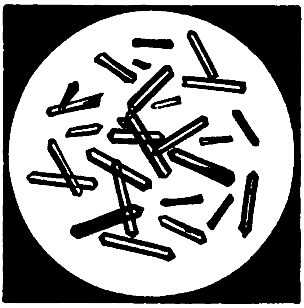
CHEMICAL ANALYSIS—ODOR OF OPIUM—PROPERTIES OF MORPHIA AND ITS SALTS —DETECTION IN ORGANIC LIQUIDS—IN THE TISSUES—DETECTION OF OPIUM AND ITS COMPOUNDS IN ORGANIC MIXTURES—TRIAL TESTS—PROPERTIES OF MECONIC ACID—NON-DETECTION OF MORPHIA AND MECONIC ACID—DIFFICULTIES IN THE ANALYSIS—RASH CHEMICAL OPINIONS.

CHEMICAL ANALYSIS.

Opium.—There are no means of detecting opium itself, either in the solid or liquid state, except by its smell and other physical properties, or by giving a portion of the suspected matter to animals, and observing the effects produced. Independently of alkaloids and principles, opium contains gum, resin, coloring matter, and other vegetable substances in variable proportion. The smell is said to be peculiar, but a similar smell is possessed by lactucarium, which contains neither meconic acid nor morphia. The *odor* is, however, a good concomitant test of the presence of the drug, whether it be in a free state or dissolved in alcohol or water, but it is not perceptible when the solution is much diluted or has been long exposed. I have found that half a grain of powdered opium, dissolved in half an ounce of water, lost its characteristic smell by a short exposure to air. The odor is decidedly volatile, and passes off when an opiate liquid is heated; it also escapes slowly at common temperatures. Again, it may be concealed by other odors, or the drug may undergo some change in the stomach during life which may rapidly destroy the odor. (Case by Mr. Barwis, *ante,* p. 537.) The analysis in cases of poisoning by opium is therefore limited to the detection of morphia and meconic acid.

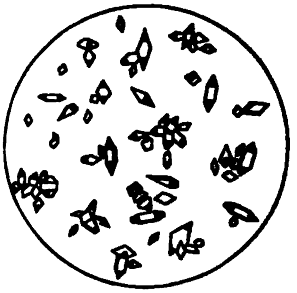
Morphia.—Morphia may be identified by the following properties: 1. It crystallizes in hexahedral prisms, which are white and perfect, according to their degree of purity (Fig. 54). These crystals may be obtained by dissolving the pure alkaloid in alcohol only, adding weak ammonia to a solution of a salt of morphia (see Fig. 55). 2. When heated on platinum, the crystals melt, become dark-colored, and burn like a resin with a yellow smoky flame, leaving a carbonaceous residue. If this experiment is performed in a small reduction-tube, it will be found, by employing test-paper, that ammonia is one of the products of decomposition. 3. Morphia is scarcely soluble in cold water; it requires 1000 parts to dissolve it, but it is dissolved by 100 parts of boiling water, and the hot solution has a faint alkaline reaction. By its insolubility in water, it is readily distinguished from its salts. It is not very soluble in ether or chloroform, thus differing from narcotina; but it is dissolved by forty parts of cold, and rather less than this quantity of boiling, alcohol. It is dissolved by a solution of potash or soda, from which it cannot be removed by ether. It is very soluble in acetic ether, and this has been employed as a substitute for ether in separating morphia from organic liquids. 4. It is easily dissolved by a very small quantity of all diluted acids, mineral and vegetable. 5. Morphia and its solutions have a bitter taste. 6. The salts of morphia are not precipitated in a crystalline form by solutions of sulphocyanide of potassium, ferrocyanide of potassium, or chromate of potash. In this respect they are strikingly distinguished from the salts of strychnia, which give well-marked crystalline precipitates with these three reagents.

FIG. 54.



Crystals of morphia from alcohol, magnified 80 diameters.

FIG. 55.



Crystals of morphia obtained by adding ammonia to a solution of the hydrochlorate, magnified 124 diameters.

Tests.—In order to apply the chemical tests for morphia, the alkaloid may be dissolved in water by the addition of a few drops of a diluted acid, either the acetic or the hydrochloric. If the hydrochlorate or the acetate of morphia is presented for analysis, the salt may be at once dissolved in a small quantity of boiling water. The tests for this alkaloid are the following: 1. *Nitric acid.* This, when added to a moderately strong solution of a salt of morphia, produces slowly a deep orange-red color. If added to the crystals of morphia or its salts, nitric oxide is evolved; the morphia is entirely dissolved, and the solution acquires instantly the deep red color above described, becoming, however, lighter by standing. In order that this result should follow, the solution of morphia must not be too much diluted, and the acid must be strong and added in rather large quantity. The color is rendered much lighter by boiling; therefore the test should never be added to a hot solution. 2. *Iodic acid* with *sulphide* of *carbon.* A solution of iodic acid should be mixed with its volume of sulphide of carbon. There should be no change of color. On adding a small quantity of these mixed liquids to morphia or its salts, either solid or in solution, the iodine is separated from the iodic acid and dissolved by the sulphide, which sinks to the bottom, acquiring a pink or red color, varying in its intensity according to the quantity of morphia present. This reaction distinguishes morphia from the other alkaloids, which do not decompose iodic acid in the cold. The fallacies connected with the use of iodic acid have been pointed out by Dr. Dupré (*Guy's Hosp. Rep.*, 1863, p. 323). The presence of morphia may be thus easily detected in one drop of the tincture of opium, in chlorodyne, nepenthe, or other opiate liquids, in spite of the presence of organic matter. If sulphide of carbon is not used, iodine may be detected by its odor or by the blue color produced on the addition of a solution of starch. 3. *Sulphomolybdic acid.* This is made by dissolving with a gentle heat eight grains of powdered molybdate of ammonia in two drachms of strong sulphuric acid. The liquid should be freshly prepared and kept from contact with air and organic matter. When one or two drops are rubbed with *dry* morphia or any of its salts, an intense reddish-purple or deep crimson color is produced. This changes to a dingy green, and ultimately to a splendid sapphire-blue. A minute trace of morphia or its salts in a solid state is thus revealed. This test produces no change in *strychnia*, but the mixture slowly acquires a pale blue tint. The presence of morphia in strychnia is thus easily detected. When poured on *brucia* this acquires a rose-red color, becoming greenish-brown, and ultimately dark blue. When mixed with *veratria*, the liquid becomes greenish-brown, and gradually passes to a dark maroon shade. The margin becomes purple, and ultimately the whole mixture acquires a deep blue color. On chloral hydrate, sulphomolybdic acid produces no change. 4. *Sulphuric acid* and *bichromate of potash.* When strong sulphuric acid is poured on pure morphia in a solid state, there is either no effect, or the alkaloid acquires a light pinkish color. On adding to this a drop of solution of bichromate of potash, or a small fragment of a crystal, it immediately becomes green (from the production of oxide of chromium), and retains this color for some time. Other alkaloids (strychnia) are not thus affected. Narcotina is turned of a bright yellow by sulphuric acid; therefore, although it becomes green when mixed with bichromate of potash, it could not be mistaken for morphia; besides, the green rapidly passes to a dingy brown color. 5. *Perchloride of iron* (sesquichloride), or colorless persulphate. Either of these solutions when saturated and neutralized (by a small quantity of potash if very acid), gives an inky blue color to a solution of morphia, or to the solid crystals. If the quantity of morphia is small, or there is much acid in the test or in the solution of morphia, the color is greenish-blue. The blue color is removed by acids, but restored by alkalies; it is also destroyed by heat; thus the iron-test should never be employed with a very acid or a very hot solution of salt of morphia. The blue color given by this test in a solution of morphia is entirely destroyed by nitric acid and replaced by the orange-red color, so that the nitric acid will act through the iron-test, but not *vice versâ.* In this way two tests may be applied to one quantity of liquid. The iron-test may be used with a solution of the alkaloid or of its salts, if unmixed with organic matter; but a small quantity of tannic or gallic acid present in a liquid, would produce a similar change of color.

Morphia and its Salts in Organic Liquids.—The liquids may be concentrated to an extract in a water-bath, and the residue treated with rectified spirit. If not already acid, a few drops of acetic acid may be added. The alcoholic liquids should be filtered, and the residue strongly pressed. This liquid is again evaporated, and the residue now treated with water feebly acidulated. A portion of it filtered may now be tested for the presence of any *alkaloidal salt* in solution by the tests already mentioned.

If the results show the presence of an alkaloid, one portion of the liquid may be submitted to dialysis, and after a sufficient time has elapsed, the dialyzed liquid may be evaporated and tested for morphia in *solution* (*ante*, p. 155).

Another portion may be concentrated, and a few drops of ammonia added. If morphia is present in sufficient quantity, crystals of that alkaloid may be slowly deposited. These should be examined by the microscope, collected, and tested for morphia in the *dry state*.

The Tissues.—The process for detecting morphia in the liver, does not differ from that adopted for the stomach and organic liquids. The organ should be sliced in small pieces, and digested in alcohol, faintly acidulated with hydrochloric or acetic acid. An aqueous solution may be afterwards obtained from the extract. I have examined the livers and tissues in several cases in which opium had been taken, and although a liquid or a residue has been obtained, which had a bitter taste, which was reddened by nitric acid, which decomposed iodic acid, or was turned green by bichromate of potash and sulphuric acid, still no alkaloid could be procured; and one or more reactions similar to those above mentioned may be obtained by an analysis of the livers of persons who have not taken opium or morphia. Either morphia may not be permanently deposited in the organs, or it may be changed in its chemical properties before it reaches them.

Opium and its compounds in Organic Mixtures.—Opium may be regarded as a complex organic solid, containing the poisonous salt, the constituents of which we wish to detect, namely, morphia and meconic acid. It is not often that in fatal cases of poisoning by opium, or its tincture, even when taken in large quantity and death is rapid, that we can succeed in detecting the alkaloid and the acid. The poison may have been removed from the stomach by vomiting, by absorption, or by active treatment, and as life is generally protracted for some hours, its entire removal is thus greatly facilitated. Surgeon-Major Ross reports that in 1869 there were forty-five cases of opium-poisoning in the Bengal Presidency, and all proved fatal. An inspection and an analysis were made in each case, but in only two out of this large number was opium found in the stomach. In the other cases there was clear evidence of poisoning from other sources. In some Indian cases, opium appears to be taken in lumps in large quantity, and if rapidly fatal it may be discovered. In a case which occurred to Dr. Penny, of Delhi, in June, 1868, in which death took place in three hours, *two ounces* of solid opium were found in the body.

Neither morphia nor meconic acid appears to be much influenced by the putrefactive process. In organic liquids containing opium, I have found them both present after the liquids had been exposed for fourteen months and allowed to undergo decomposition spontaneously in the air.

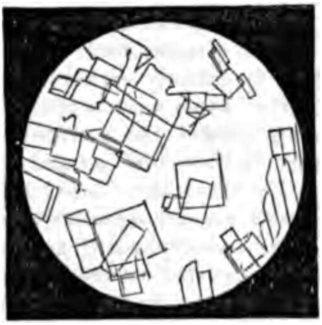
Meconic acid is more commonly and more easily discovered than morphia, and its discovery has this importance, that there is no substance but opium in which this acid has yet been detected, and the test for it is more reliable and less open to objection than any test which can be applied to morphia. The alkaloid and the acid may in general be detected more readily in the matter vomited during life (if vomiting should have occurred) than in the contents of the stomach after death. It was thus detected in the form of aqueous infusion in a case where the contents of the stomach had been ejected about seven hours after the poison had been swallowed. (*Medical Gaz.*, vol. 37, p. 724.) Before resorting to any process for the separation of morphia or meconic acid from an opiate compound contained in a suspected liquid, it will be necessary to employ trial tests to determine whether any appreciable quantity of either is present. The *smell* of opium has already been mentioned as a rough test of the presence of the drug; but this may not be perceptible, and yet the acid and the alkaloid may still be present.

A small portion of the organic liquid, poured off clear, or filtered, should be well shaken with a solution of iodic acid and its bulk of sulphide of carbon. The mixture is allowed to stand in a corked tube. The sulphide after a time falls to the bottom, and is colored more or less pink (from dissolved iodine), if any morphia is present. Morphia may be thus detected in chlorodyne, paregoric, and other opiate preparations. Any alcohol present should be first distilled off. This result is presumptive but not conclusive of the presence of morphia, and therefore of opium, in the liquid. Thus saliva (owing to its containing an alkaline sulphocyanide), an infusion of white mustard, and a solution of a sulphocyanide, or any deoxidizer, will produce a similar result. Strong nitric acid added to a portion of the organic liquid, sufficiently diluted to allow a change of color to be perceived, will, however, in many cases, serve to corroborate the results of the iodic acid test.

If mixed with much foreign matter, it will be better to pass a portion of the organic liquid through the tube dialyzer (p. 154, *ante*), and then apply the iodic and nitric acids to the dialyzed liquid.

Meconic Acid.—This is a solid crystalline acid, seen commonly in scaly rectangular crystals of a pale reddish color. It is combined with morphia in opium, of which, according to Mulder, it forms on an average six per cent.; and it serves to render this alkaloid soluble in water and other menstrua. *Tests.*—Many tests have been proposed for meconic acid; there is only one upon which any reliance can be placed, namely, the *perchloride* or *persulphate of iron.* This test produces, even in a diluted solution of meconic acid, a deep blood-red color; and it is owing to the presence of this acid that a salt of iron strikes a red color in tincture and infusion of opium, as well as in all liquids containing traces of meconate of morphia, the effects of the iron-test with morphia being counteracted by the presence of meconic acid. The red color of the meconate of iron is not destroyed by diluted sulphuric acid or by a solution

FIG. 56.



Crystals of meconic acid, magnified 70 diameters.

of corrosive sublimate. To a portion of the diluted suspected liquid, a persalt of iron should therefore be added. If this strikes a red color which is not discharged by boiling with dilute sulphuric acid, it indicates the presence of *meconic acid*. In liquids containing tannic acid, *e. g.*, tea or beer, the action of this test is obscured by the production of tannate of iron. The dark color is removed by a few drops of diluted sulphuric acid, and the red tint then appears.

It is a remarkable fact that healthy *saliva* contains a substance which, like morphia, decomposes iodic acid (and produces a blue color with a solution of starch), while at the same time it gives a deep red color resembling that produced by a persalt of iron in meconic acid. This source of error in analysis was first brought to the knowledge of the profession by the evidence given at a trial in Edinburgh (case of Stewart, 1829), in which the late Dr. Ure was cross-examined for the accused. An alkaline sulphocyanide and an acetate in solution produce a similar red color with the iron-test; but, unlike the meconate, this color is destroyed by heating the liquid with two or three drops of diluted sulphuric acid.

If the trial tests thus indicate in the supposed opiate mixture, the presence of morphia and meconic acid, the next step will be to separate these substances from the organic liquid.

If the matter is solid, it should be cut into small slices; if liquid, evaporated in a water-bath to an extract; and in either case digested with alcohol and a small quantity of diluted acetic acid for one or two hours at a gentle heat. The liquid portion may be strained off, pressed, and again brought to an extract in a water-bath. It may now be digested in distilled water acidulated, until all the soluble matter is taken up. The aqueous acid solution thus obtained may be concentrated and treated with a solution of acetate of lead until a precipitate is no longer produced. The liquid with the precipitate should be boiled and filtered; meconate of lead is left on the filter, while any morphia passes through under the form of acetate. The surplus acetate of lead in the filtered liquid (containing the morphia) should now be precipitated by a current of sulphuretted hydrogen gas, the sulphide of lead separated by filtration, and the liquid evaporated at a gentle heat, in a water-bath, to an extract, so that any sulphuretted hydrogen may be entirely expelled. The sulphide of lead which is precipitated carries down with it much organic matter. On treating this extract with alcohol, the acetate of *morphia*, if present in sufficient quantity, may be dissolved out and tested.

The *meconate* of lead left on the filter may be decomposed by boiling it with a small quantity of diluted sulphuric acid; and in the filtered liquid (neutralized if necessary by an alkali) the meconic acid may be easily detected by the iron-test. This analysis requires care as well as some practice in the operator, in order that the morphia should be obtained in a sufficiently pure state for the application of the tests, but the quantity present in the liquid may really be too small for separation by this process. Other methods of separation have been suggested, but there is none so effectual in obtaining meconic acid as that above described. Dr. Wormley has found that by this process he was able to procure evidence of the presence of meconic acid and morphia from a complex organic mixture containing only one grain of opium. (*Micro-Chemistry of Poisons*, p. 497.) This would represent about the tenth part of a grain of morphia, and the sixteenth part of a grain of meconic acid.

Another method has been recommended. Thus, instead of precipitating the concentrated aqueous solution of the alcoholic extract with acetate of lead, a small quantity of ammonia is added, and the liquid is allowed to stand. Meconate of ammonia is produced in the liquid, and after a time morphia is deposited in a crystalline or amorphous state. If the deposit is much colored, it should be dissolved in a minimum quantity of hot water, barely acidulated with hydrochloric acid, and set aside to crystallize. On cooling the liquid, prisms of hydrochlorate of morphia, sufficiently pure for testing, may sometimes be procured. If no crystals are deposited, the liquid should be still further concentrated, either by spontaneous evaporation, or in a water-bath. If again no crystals are formed, ammonia may be added, and the precipitate (if any) may then be tested. It is better for the purposes of testing to procure a small quantity of morphia in a pure state, than to have a large quantity in an impure state. A very elaborate process, based on this method of separation by ammonia, has been described by MM. Tardieu and Roussin. (*L'Empoisonnement*, 1867, p. 891.) It consists of twelve different stages. Tartaric acid is employed in place of the acetic, and nearly absolute alcohol in place of rectified spirit. They advise that morphia should be obtained in the solid state, and the tests selected for the alkaloid should be nitric acid, a persalt of iron, and iodic acid. (*Op. cit.*, p. 894.)

The object of this process appears to be rather to isolate the morphia than the meconic acid. In order to demonstrate the presence of the latter, the liquid remaining after the precipitation by ammonia—consisting of meconate of ammonia—should be slightly acidulated with hydrochloric acid, and then treated with a few drops of a persalt of iron. This brings out the deep-red color characteristic of the meconate of iron. (*Op. cit.*, p. 895.)

From the small proportion of morphia (p. 528) and meconic acid (six per cent.) contained in opium, it is obvious that unless the soluble matter of one or more grains of opium is present, it will not be easy to separate any morphia or meconic acid from an organic liquid. If the quantity of opium be less than half a grain, acetate of lead will not precipitate any perceptible quantity of meconate of lead, and it would require from one-quarter to half a grain of dry meconate of lead to procure good evidence of the presence of meconic acid. When the quantity of meconate of lead is small, it should be placed in a watch-glass, covered with a few drops of diluted sulphuric acid, and gently warmed. Having allowed time for the subsidence of sulphate of lead, the persalt of iron may be added, and the change of color noticed.

These facts will explain why the constituents of opium are so seldom found in the contents of the stomach after death, especially in the cases of infants or children killed by this drug. In the case of a young woman who died *five hours* after taking *two ounces* of laudanum, Sir R.

Christison did not succeed in detecting morphia by any of the tests. Other cases of a similar kind are mentioned by him. In several instances of poisoning with opium which have occurred to myself, there has not been a trace of meconic acid or of morphia in the contents of the stomach. In one instance, a woman swallowed an ounce and a half of laudanum in beer. In half an hour she was in a state of profound coma, and she died in nine hours. None of the poison could be detected in the stomach; there was not even the smell of opium. In two cases, which occurred in 1844, one having proved fatal in five, and the other in twenty-two hours, there was not the least trace of opium either by the odor, or by tests. In the latter case half an ounce of the tincture had been taken. The cause of the failure of chemical evidence on these occasions is partly due to the smallness of the quantity which may remain in the body at the time of death, and partly to its entire removal by vomiting, absorption, or digestion. Infants have been killed by doses of opium equivalent to less than the hundredth part of a grain of morphia (p. 542). To suppose that any portion of morphia or meconic acid could be separated after death from the body of a child that had survived some hours would be absurd; yet some chemists, calling themselves "toxicological experts," have shamelessly attempted to deceive the public by swearing that no person could die of poison, except the poison remained in, and was visibly separable from, the stomach or tissues after death!

As there is no medicine so frequently prescribed as opium in ordinary disease, an analyst must remember that the discovery of a small quantity in the stomach is not sufficient to establish the fact of poisoning. It may be the residuary quantity of an opiate medicine lawfully prescribed for the deceased.

It need hardly be observed that, except for a preliminary trial, the tests for morphia should not be applied to liquids which contain organic matter. A decoction of mustard-seed produces with two of the tests—the nitric and iodic acids—changes similar to those produced by morphia; while with a persalt of iron it produces a red color resembling that caused by meconic acid. An infusion or decoction of nux vomica is reddened by nitric acid, and there are many common vegetable infusions and decoctions (pimento, cloves, senna, etc.), which acquire a red or orange color when nitric acid is added to them. Again, the persalts of iron give a bluish color with tea, beer, and numerous organic liquids containing gallic or tannic acid. Iodic acid is decomposed by a still larger number of substances, including putrescent animal or vegetable matter, and all deoxidizing agents. Owing to an undue reliance upon the tests, as applied to organic liquids, some serious mistakes have been already made. Thus, in the case of Major Forester, on whose body an inquest was held in 1852 (*Legal Examiner*, Oct. 9, 1852), an "analytical chemist" who gave evidence, deposed to the presence of morphia in the rectum of the deceased, as well as in the urine and the blood, on the ground chiefly that sulphuric acid and bichromate of potash produced a green color! He appears to have been utterly ignorant that sugar and a large number of organic substances will produce a similar color by setting free oxide of chromium when mixed with sulphuric acid and bichromate of potash; and on this fallacious mode of testing, he confidently swore that he found the two-thousandth part of a grain of morphia in the blood!

Under a like degree of rashness, traces of morphia have been sworn to be present in the stomach of a person dying of ordinary disease, in cases in which there was not the slightest evidence of possible administration, and a total absence of any symptoms during life. (See *ante*, pp. 123, 152.) These rash opinions, based on the alleged presence of a quantity of an alkaloid too small to be seen or weighed, have had the effect of damaging the reputation of medical men or of relatives who attended the deceased. It is only recently that a lady, the wife of a medical gentleman, has been driven to an act of suicide by such a charge being thus made against her. If coroners and lawyers were better acquainted with scientific processes, such testimony would be at once rejected. (*Guy's Hosp. Rep.*, 1874, p. 467.)

CHAPTER LIX.

PRUSSIC ACID—EFFECTS OF THE VAPOR—SYMPTOMS—TASTE AND ODOR OF THE ACID—PERIOD AT WHICH SYMPTOMS COMMENCE—LOSS OF CONSCIOUSNESS AND MUSCULAR POWER—EFFECTS CONTRASTED WITH THOSE OF OPIUM—CHRONIC POISONING—EXTERNAL APPLICATION—APPEARANCES AFTER DEATH—FATAL DOSE—TREATMENT.

PRUSSIC or HYDROCYANIC ACID, owing to its rapid and unerring effects when taken even in comparatively small doses, is one of the most formidable poisons with which we are acquainted. The pure or anhydrous acid requires no notice here, since it is not likely to be met with out of a chemical laboratory. The common acid is a mixture of this pure acid with water, and sometimes with alcohol. As it is sold in shops, it varies in strength. I have found different specimens to contain from 1.3 to 6.5 per cent. of the strong acid; but two varieties are now commonly met with: 1. The prussic acid of the British Pharmacopœia, containing about two per cent.; and, 2. Scheele's acid, containing from four to five per cent. Owing to the great volatility of prussic acid, it is rare to find two samples of uniform strength. In a case of poisoning which was referred to me, in 1847, an acid sold as Scheele's was found to contain only two per cent. (*Med. Gaz.*, vol. 40, p. 171.) The pharmacopœial acid rarely holds its due proportion. It sometimes exceeds and sometimes falls below two per cent. (*Pharm. Jour.*, Sept. 1874, p. 192.) This variation seriously affects the medicinal and poisonous doses of the two acids. The stronger the acid the greater the proportionate loss by use and exposure. A much weaker preparation of this acid should alone be used for medicinal purposes. The *medicinal dose* of Scheele's acid is from a minim to two minims; of the British pharmacopœial acid, from two to eight minims, gradually increased. On the Continent, the acid is met with of a strength rising as high as from ten to twenty-five per cent. The following is the percentage strength in anhydrous acid of the different varieties in *aqueous* solution. Acid of Schrader (acid of the Prussian Pharmacopœia), 1; British and United States Pharmacopœia, 2; Göbel, 2.5; Vauquelin and Giese, 3.3; Scheele, 4 to 5; Ittner, 10; Robiquet, 50. Among the *alcoholic* solutions of the acid: Schrader, 1.5; Bavarian Pharmacopœia, 4; Duflos, 9; Pfaff, 10; Keller, 25 per cent. The price at which the acid is sold to the public is about two shillings an ounce.

Poisoning by prussic acid is frequently the result of suicide or accident. In 1837-8 there were twenty-seven cases of poisoning by this liquid, nearly all of which were the result of suicide. Of late years it has, however, acquired a fatal celebrity as a means of murder! Several murders have been perpetrated and several attempted by this poison. In five years, 1863-7, there were 151 fatal cases of poisoning with prussic acid and cyanide of potassium.

Prussic Acid Vapor.—The vapor of anhydrous prussic acid, if respired, would prove almost instantaneously mortal. Even the vapor of the diluted acid accidentally respired may occasion serious symptoms. A medical practitioner, while showing to some friends the effects of Scheele's prussic acid on an animal, accidentally allowed a quantity of the acid to fall upon the dress of a lady who was standing before a fire. The poison was rapidly evaporated, and the lady was immediately seized with dizziness, stupor, inability to stand, and faintness. The pulse was feeble and irregular. Brandy was administered, cold affusion employed, and the patient was exposed to a free current of air. In ten minutes the pulse began to improve, and with the exception of trembling in the limbs, the unpleasant symptoms disappeared. I have known headache and giddiness produced by the vapor from the small quantities used in ordinary chemical experiments. Some caution is required even in smelling a bottle containing a strong specimen of this acid. Chemical experiments show that this poison is always in the act of escaping from liquids which contain it; and the quantity evolved and diffused, depends on temperature and the surface of liquid exposed to air.

I am not aware that there is any well-authenticated case of death having been caused by the vapor. The celebrated Scheele died suddenly while making his researches on this poison, and it is alleged that he was killed by breathing the vapor of the diluted acid. In October, 1847, a question arose at an inquest in this metropolis whether the vapor of Scheele's acid had caused death. The deceased entered a druggist's shop, and requested to be shown a bottle of Scheele's prussic acid. He suddenly attempted to snatch the bottle from the hand of the assistant: a struggle ensued, during which a portion of the acid was spilled over the deceased's face, and over the coat of the assistant. The deceased ran into a neighboring shop, and died in about a quarter of an hour. At the inquest it was alleged that death had been caused by the vapor, owing to the acid having been spilled over the deceased's face. Of this, however, there was no proof, as the body was not inspected for the inquest! It is most probable that the deceased had swallowed a sufficient quantity of the acid to cause death.

Dr. Regnauld has reported a case in which a student nearly lost his life by respiring the vapor of prussic acid as it escaped from a flask in which he was preparing the poison. He lay in a perfectly insensible state for many hours. There was slight lividity of the face, the eyelids were closed, and the pupils were widely dilated; the breathing was difficult, and took place at intervals. The limbs were cold, and the pulse was barely perceptible. The muscles of the arms and legs were firmly contracted, but there were no tetanic convulsions. The patient was in a state of complete coma, and could not be roused. After some hours, recovery took place, but he suffered from headache and other symptoms. (*Ann. d'Hyg.*, 1852, vol. 1, p. 455.) From these facts there is no doubt that the concentrated vapor, if breathed, would speedily destroy life.

Dr. Chanet has directed attention to the effects slowly produced by prussic acid vapor upon those who breathe it when diffused in a very diluted state. The process of galvanic gilding and silvering is now very common. Cyanide of potassium is used as a solvent for the metals, and as the solution is freely exposed to the air prussic acid is always passing off in vapor from its surface. The evolution of the vapor is aided by warmth, and its noxious effects are aggravated by the closeness and want of ventilation in the rooms in which the process is carried on. The whole manufactory is perceptibly infected with the odor, and the workmen are thus compelled to breathe a poisonous atmosphere for many hours together. Dr. Chanet satisfied himself respecting the diffusion of the acid, by placing above the cyanide-bath a solution of nitrate of silver. A white film of cyanide of silver was immediately produced on the surface. Some of the men are obliged to abandon the work from a feeling of illness. The symptoms among those who remain for a long time exposed to the vapor are dull headache, accompanied by shooting pains in the forehead, noises in the ears, giddiness, dizziness, and other effects indicative of cerebral congestion. Then follow difficult respiration, pain in the region of the heart, a sense of suffocation, constriction in the throat, and palpitation, with alternate fits of weakness and somnolency. (*Gazette des Hôpitaux*, 24 Juillet, 1847.) In trying some experiments on galvanic gilding, a few years since, I found that the evolution of the prussic acid vapor was so manifest that a solution of nitrate of silver was whitened when exposed in the apartment at some distance, the whole apparatus was therefore kept covered over.

Symptoms.—This acid in large doses is described as having a hot, bitter taste, and an odor resembling that of bitter almonds diluted. The time at which the symptoms of poisoning commence in the human subject is liable to great variation from circumstances not well understood. When a large dose has been taken, as from half an ounce to an ounce of the diluted acid, the symptoms usually commence in the act of swallowing, or within a few seconds. It is rare that their appearance is delayed beyond *one or two minutes*. When the patient has been seen at this stage he has been perfectly insensible, the eyes fixed and glistening, the pupils dilated and unaffected by light, the limbs flaccid, the skin cold and covered with a clammy perspiration; there is convulsive breathing at long intervals, and the patient appears dead in the intermediate periods; the pulse is imperceptible, and involuntary evacuations are occasionally passed. The respiration is slow, deep, gasping, and sometimes heaving, or sobbing. The following case, communicated to me by Mr. French, presents a fair example of the immediate effects of this poison in a large and fatal dose: A medical man swallowed seven drachms of the common prussic acid. He survived about four or five minutes, but was quite insensible when discovered, *i. e.*, about two minutes after he had taken the poison. Mr. French was called to him immediately, and found him lying on the floor senseless. There were no convulsions of the limbs or trunk, but a faint flickering motion was observed about the muscles of the lips. The breathing appeared to cease entirely for some seconds; it was then performed in convulsive fits, and the act of respiration was remarkably deep, and lasted for an unusual time.

When the dose is large, the breath commonly exhales a strong odor of the acid, and this is also perceptible in the room. Convulsions of the limbs and trunk, with spasmodic closure of the jaws, are usually met with among the symptoms; the finger-nails have been found of a livid color, and the hands firmly clenched. The breathing is generally convulsive, but when the coma or insensibility is profound, it is sometimes stertorous. This was noticed in a case which occurred to Sir R. Christison. (*Edinburgh Monthly Journal*, February, 1850, p. 97.) It was also observed in the case of Marcooley (*Reg.* v. *Boroughs*, C. C. C., February, 1857). Stertorous breathing has not been recorded by toxicologists as one of the usual symptoms of poisoning by prussic acid. In the inquiry which took place at Rugeley, in January, 1856, respecting the death of Walter Palmer, it was contended that the fact of the deceased having had stertorous breathing was a proof that he had died from apoplexy, and not, as it was alleged, from prussic acid; but the facts here recorded show that such an inference is not justified by experience.

When a small dose (*i. e.*, about thirty minims of a weak acid) has been taken, the person has first felt a sense of weight and pain in the head, with confusion of intellect, giddiness, nausea, a quick pulse, and loss of muscular power; these symptoms are, however, sometimes slow in appearing. Vomiting has been occasionally observed, but it is more common to find foaming or frothing at the mouth, with suffusion or a bloated appearance of the face, and prominence of the eyes. If death results, this is preceded by tetanic spasms, opisthotonos, and involuntary evacuations. Vomiting is sometimes the precursor of recovery. (See cases in *Medical Gazette*, vol. 36, p. 103; vol. 35, pp. 859, 893.) A case which occurred to Mr. Bishop (*Prov. Med. and Surg. Jour.*, Aug. 13, 1845, p. 517) was remarkable in several particulars; the man swallowed, it was supposed, forty minims of an acid (at three and a quarter per cent.), and was able to give an account of his symptoms. He was conscious for some time after he had taken it, and he recollected experiencing the sensation of his jaws becoming gradually stiff and tight against his will.

Exercise of Volition and Locomotion.—One of the most marked effects of prussic acid is to produce insensibility and loss of muscular power much more rapidly than any other poison. In some instances, there may be loss of consciousness in a few seconds; in others, certain acts indicative of volition and locomotion may be performed, although requiring for their performance several minutes. This is one of the most important questions connected with death from prussic acid. In treating this subject, Dr. Lonsdale says that a drachm of Scheele's acid would affect an ordinary adult within a *minute;* and if the dose were three or four drachms, it would exert its influence within ten or fifteen seconds. When the acid is stronger and the quantity larger, we are pretty certain of its immediate action, and the consequent annihilation of the sensorial functions. (*Ed. Med. and Surg. Jour.*, vol. 51, p. 50.) A case was communicated to me by one of my pupils, where a man was found dead on the seat of a water-closet; he had died from prussic acid, and the bottle which had contained the poison was in his pocket, corked. Many similar facts are recorded which show that while, as a general rule, insensibility may supervene from a large dose of this poison in a few seconds, a person may occasionally retain for one or two minutes a power of performing certain acts indicative of consciousness, volition, and locomotion. In the case of Mr. Burman, a medical man, sensibility, consciousness, and a power of swallowing were retained for *two minutes*, after a large dose of prussic acid had been swallowed. (*Lancet*, Jan. 14, 1854.) The importance of this question may be judged of by its bearing on the case of *Rex* v. *Freeman*, which was tried at the Leicester Spring Assizes, 1829. (*Medical Gazette*, vol. 8, p. 759.) A young man, named Freeman, was charged with the murder of Judith Buswell, by administering to her prussic acid. The deceased was one morning found dead in her bed; her death had been evidently caused by prussic acid, and it was presumed that she had taken four and a half drachms of Scheele's acid; the bottle out of which she must have drunk it, or had it administered to her, held an ounce, and it contained when found three and a half drachms. Owing to the position of the body when discovered, and other circumstances connected with it, it was inferred that she could not have taken the poison herself. Her body was lying at length on the bed, the head being a little on one side. The bed-clothes were pulled up straight and smooth, and they came up to her breast; her arms were under the clothes, and crossed over the chest. On turning the clothes aside, the vial which contained the poison was found lying on her right side. It was corked, and there was a piece of white paper round it; the leather and string which appeared to have gone round the neck of the bottle were in the chamber-vessel. The medical question at the trial was—Could this quantity of poison have been taken, and the deceased have retained volition and consciousness for a sufficiently long period to have performed these acts herself? Five medical witnesses were examined, and the opinions of four of these were strongly against the *possibility* of the acts having been performed by the deceased. This strong medical opinion was, however, completely set aside by circumstances, and the prisoner was acquitted. All the acts to which the opinion referred, might be performed in from *five to eight seconds;* and there was nothing to justify the witnesses in asserting that under the above-named dose, all power would necessarily have ceased before this short period of time had elapsed. A similar case, quoted by Sobernheim, has since occurred in Germany. A young man swallowed four ounces of an acid (of four per cent.), equivalent to eight ounces of the pharmacopœial strength! He was found dead in bed, the clothes drawn up to the breast, the right arm stretched out straight beneath the clothes, the left bent at the elbow-joint, and on each side of the bed lay an empty two-ounce vial. There was no doubt of this being an act of suicide. In this case more than three times as much acid was taken as in that of Buswell, but even here there was time for the performance of similar acts! It is besides much more difficult to understand how the poison should have been taken out of two vials than out of one. Some years since I was required to examine a case of suicide by prussic acid in which the facts were strongly confirmatory of the views here expressed. The deceased swallowed *three drachms* of prussic acid, and was found dead in bed, the clothes being smoothly drawn up to his shoulders, and there was no appearance of disorder about them, nor was there any sign of struggling before death. On a chair at the back of the bed, but close to it, was the vial which had contained the prussic acid with the cork in it. (*Guy's Hosp. Reports*, April, 1845.) There could not be the slightest doubt that the deceased had committed suicide, and that, after swallowing the poison, he had retained sufficient sense and power to perform these acts. (See also other cases, *Lancet*, September, 1874; and June 7, 1845.) In death from prussic acid the body is usually found lying calm and tranquil, without any mark of struggling or convulsions.

Locomotion and muscular exertion are, of course, compatible with small but fatal doses of this poison. In a case which occurred to Mr. Hicks, a girl sprang from her seat after swallowing a small dose of acid, threw her arms over her head, gasped for breath, and ran forwards about two yards, before she fell. In one, reported by Mr. T. Taylor, the man ran twelve or fourteen paces before he fell, and remained insensible for a space of four hours—a very long duration for the effects of this poison without causing death. Other cases have shown that prussic acid does not give rise to insensibility and other alarming symptoms so speedily as it was formerly supposed. Mr. Garson, of Stromness, has reported an instance in which a person, for medicinal purposes, took at least a teaspoonful of prussic acid (the strength not mentioned); the symptoms, however, did not come on for a *quarter of an hour*, when the patient was found insensible. He recovered, and stated that that period of time had probably elapsed between the taking of the dose and the commencement of the symptoms, and that he had employed himself in writing during the intermediate period! (*Ed. Med. and Surg. Jour.*, vol. 59, p. 72.) See also a case by Mr. Godfrey (*Prov. Med. Jour.*, Sept. 25, 1844; *Med. Gaz.*, vol. 40, p. 171; and *Guy's Hosp. Rep.*, Oct. 1846, p. 490.) The facts accumulated in reference to this question are now so numerous and well authenticated, that it is never likely to become again a subject of doubt or dispute in a court of law, or to place the life of an accused person in jeopardy.

If we contrast the effects of this poison with those of opium, we shall find the following general differences. In opium, the coma comes on gradually, and is seldom seen until after the lapse of a quarter of an hour. In poisoning by prussic acid, coma is rapidly induced; even in weak doses, insufficient to prove fatal, this symptom is rarely delayed beyond two minutes. Convulsions may be met with in both forms of poisoning, but perhaps more commonly in poisoning by prussic acid. With respect to the occurrence of this symptom, it is a fair question whether medical jurists have not too readily adopted views from the results of experiments made on animals, not from observations on man; since in very few instances, when the dose of poison has been *large*, has the patient been seen alive. When the dose was small, but still fatal, convulsions have been sometimes observed. In poisoning by opium the pupils are contracted, except in the advanced stage, when they may be found dilated. In poisoning by prussic acid they are commonly dilated. A well-marked remission or intermission of the cerebral symptoms has been frequently noticed before death, in poisoning with opium; this has not been witnessed in poisoning with prussic acid: the symptoms once produced have been observed to progress in severity until death. In poisoning with prussic acid, the case, if fatal, generally terminates in less than an hour; in poisoning by opium, the average period of death is in from six to twelve hours. The time at which the symptoms appear after a liquid has been swallowed, their sudden invasion, the almost immediate loss of sensibility, and the odor of the breath, would, under ordinary circumstances, suffice to establish a clear distinction between the effects of the two poisons.

A question has arisen, whether prussic acid is an accumulative poison; *i. e.*, whether, after having been taken in small doses and at short intervals without apparent mischief, it may by accumulating in the blood suddenly give rise to all the effects of poisoning, either on a repetition of the same dose, or by a slight increase in quantity. (*Ed. Med. and Surg. Jour.*, vol. 51, p. 49.) There are no facts to support this theory. The cases are explicable on the supposition that medicinal doses have been given at intervals too short for complete elimination.

Effects of External Application.—Prussic acid is said to act through a wounded portion of skin. Sobernheim mentions the case of an apothecary at Vienna, who died in an hour from the entrance of the poison into a wound in the hand, produced by the breaking of a glass vessel in which it was contained. It is also said to act through the unbroken skin; but this does not appear to be the case with the common diluted acid. The acid would doubtless produce all the effects of poisoning, if applied to an ulcerated or any highly absorbing surface. It has been found, in experiments on animals, that the poison acts with the same rapidity and certainty on applying it to the mucous membrane of the conjunctiva, rectum, or vagina, as when swallowed. (*Prov. Trans.*, N. S. vol. 3, p. 84.)

Appearances after Death.—The body when seen after death often exhales the odor of prussic acid; but if it has remained exposed before it is seen, and if it has been exposed to the open air or in a shower of rain, the odor may not be perceptible; again, the odor may be concealed by tobacco-smoke, peppermint, or other powerful odors. In a case in which a person poisoned himself with two ounces of the acid, and his body was examined twenty-eight hours after death, the vapor of prussic acid which escaped on opening the stomach was so powerful that the inspectors were seized with dizziness. In cases of suicide or accident, the vessel out of which the poison had been taken will commonly be found near; but there is nothing to preclude the possibility of a person throwing it from him in the last act of life, or even concealing it if the symptoms should be delayed. (See case by Christison, p. 298.) Owing to the great volatility of the poison, the vessel, if left uncorked, may not retain the odor when found. Putrefaction is said to be accelerated in these cases; but from what I have been able to collect, there seems to be no ground for this opinion. (*Prov. Med. Jour.*, July 30, 1845.)

Externally, the skin is commonly livid, or is tinged of a violet color; the nails are blue, the fingers clenched, and the toes contracted; the jaws firmly closed, with foam or froth about the mouth, the face often pallid, but sometimes bloated and swollen, and the eyes have been observed to be wide open, fixed, glassy, very prominent and glistening, and the pupils dilated; but a similar condition of the eyes has been observed in other kinds of violent death. *Internally*, the venous system is gorged with dark-colored liquid blood; the stomach and intestines may be in their natural state; but in several instances they have been found more or less congested. The mucous membrane of the stomach of a dog, which died in a few minutes from a dose of three drachms of Scheele's acid, was intensely reddened throughout, presenting the appearance met with in cases of arsenical poisoning. In a large number of experiments upon dogs, the late Mr. Nunneley found generally a congested condition of the mucous membrane of the stomach; if empty at the time the poison was taken, this organ was found much contracted, and of a brick-red color. This appearance of congestion was observed on the mucous membrane of the vagina, the rectum, and conjunctiva, when the acid was applied to these parts. (*Prov. Trans.*, N. S., vol. 3, p. 79.) Redness of the stomach was noticed in the cases of the Parisian epileptics. (*Ann. d'Hyg.*, 1829, vol. 1, p. 507; also in a case which occurred at Guy's Hospital under Dr. Hodgkin.) The late Dr. Geoghegan, of Dublin, communicated to me the particulars of a case in which the redness of the mucous membrane was well marked. In April, 1847, a healthy man, æt. 30, swallowed a large dose of prussic acid. He was afterwards found dead in his bed. The body was inspected in five hours; rigidity had then commenced, and there was some warmth. The face was pale, the eyes were half closed, not presenting any remarkable brilliancy or prominence, and there was great dilatation of the pupils. The mouth was closed, and no froth issued from it. The abdomen was the only cavity examined. The muscles were red, and gave out, on section, a good deal of fluid blood, which had a strong odor of prussic acid; the odor of the poison was also perceptible in the abdomen. About eight ounces of a thick farinaceous mass were found in the stomach; the odor of prussic acid was very perceptible in this organ, but it was mixed with that of rancid food. The mucous membrane had everywhere, except at the greater end and posterior wall, a vivid inflammatory redness of a well-marked character, and it was covered with a layer of viscid mucus to a considerable extent. This membrane, even after it had been washed three times in water, gave out a strong odor of prussic acid. In a case which I examined in May, 1850, in which death had been caused by a large dose of the acid, there was a generally congested state of the mucous membrane of the stomach. I am indebted to Mr. Baker, of Lewes, for an account of the appearances in the body of a medical student who destroyed himself in March, 1860, by swallowing about one drachm of Scheele's acid. He was found in a state of collapse and breathing heavily, in about half a minute from the time at which he was last seen. He died in twenty minutes. The coats of the stomach were greatly congested towards the cardiac end. The minute vessels throughout were filled with dark blood, and there were some spots of effused blood beneath the mucous coat. The intestines were highly congested, the small vessels being visible all over the coats. There was no congestion of the membranes of the brain. Dr. Frank has recorded the appearances in two cases which fell under his notice. (Horn's *Vierteljahrsschrift*, 1868, vol. 2, p. 179.)

In a case which occurred to Casper, the liver, kidneys, and the large veins of the abdomen and chest, as well as the lungs, were filled with dark fluid blood; there was a small quantity in the right ventricle, while the brain and its membranes were bloodless. There were red patches on the stomach and œsophagus. (Casper, *Ger. Med.*, vol. 1, p. 432.) Congestion of the brain and its membranes, of the lungs, heart, mucous membrane of the stomach and bowels, of the liver, spleen, and kidneys, have been found more or less in different cases, but there is no constancy or uniformity in the appearances; and, but for the presence of the well-known odor of the poison, there would have been in some cases no suspicion of the cause of death. The *odor* of the poison, if not observed in the body, is generally perceptible in the stomach for several days after death, unless the quantity of poison is small, and it is mixed up with other strongly smelling substances. If death has been rapid, the dose large, and the inspection recent, as in the case just related, all the cavities, as well as the blood, have the odor.

Besides the appearances above described, the brain and lungs have been found congested, although not invariably. The *blood* is, in some instances, quite liquid, in others thick and semi-coagulated. (Heller's *Archiv.*, vols. 1, 2, 1845, p. 143.) In most cases this liquid has been found of a very dark color, in a few red, and in other cases again of a violet or pinkish hue.

After this description of the appearances met with in death from a large dose of the poison, it may be proper to state those found in the body of a woman killed by the smallest dose of prussic acid yet known to have destroyed life—nine-tenths of a grain. The inspection was made ninety hours after death. The teeth were clenched, and some froth was still adhering to the mouth; the face was of a dusky red hue, and the whole of the depending part of the body of a dark purple or violet color; it had very much the appearance of the body of a person who had died from asphyxia. The dura mater and sinuses were much congested, and the whole of the substance of the brain was dotted with blood, which was fluid and very black; the ventricles were empty, and the plexus choroides pale and bloodless; but no odor of prussic acid was perceptible. On opening the chest, the odor was more plainly perceived than in any other part of the body; the lungs were much congested, but otherwise healthy; the right ventricle of the heart was distended with fluid black blood. The stomach contained four ounces of a liquid smelling strongly of prussic acid; its lining membrane was healthy, with the exception of a small patch of redness near the cardiac orifice; but, as the deceased had suffered from gastric symptoms, this may have been owing to disease, and not to the action of the poison. The liver, gall-bladder, and kidneys were healthy, except that the latter were congested, and had a dark pinkish hue. (*Med. Gaz.*, vol. 36, p. 460.)

Fatal Dose.—The *smallest* dose of this acid which is reported to have caused death, was in a case which occurred to Mr. Hicks. (*Med. Gaz.*, vol. 35, p. 896.) A healthy adult woman died in twenty minutes from a dose equivalent to *nine-tenths* of a grain of anhydrous prussic acid. This was equivalent to about *twenty grains* of Scheele's acid. In a case reported by Mr. T. Taylor (*Med. Gaz.*, vol. 36, p. 104) a stout healthy man swallowed this dose, *i. e.*, nine-tenths of a grain, by mistake, and remained insensible for *four hours*, when he vomited, and began to recover. The vomited matters had *no odor* of the poison, showing that, if not concealed by other odors, the whole of the acid must have been absorbed. He had a very narrow escape of his life. Dr. Banks has published a case in which a woman recovered after swallowing thirty drops of prussic acid. (*Ed. Med. and Surg. Jour.*, vol. 48, p. 44.) The *largest* dose from which an adult has recovered, was probably in a case which has been reported by Mr. Burman. (*Lancet*, Jan. 14, 1854.) His father, æt. 60, of a strong constitution, took by mistake a *drachm* of prussic acid, equivalent to 2.4 grains of anhydrous acid. In a few seconds he perceived the mistake, and swallowed half an ounce of aromatic spirits of ammonia with a little water. Four minutes after taking the poison, cold affusion was employed, and sulphate of iron and spirit of ammonia were administered. Vomiting, with convulsive shuddering and insensibility, took place. In twenty minutes consciousness returned, and fifteen minutes later he was able to walk upstairs to bed. He perfectly recovered, but in the absence of the early treatment resorted to, it is most probable that he would have died. Sir R. Christison has reported in the *Edinburgh Monthly Journal* (Feb. 1850, p. 97), the case of an adult who recovered after taking a dose equivalent to a *grain and a half* or two grains of anhydrous acid. The treatment consisted in the evacuation of the stomach by the stomach-pump, and in pouring a current of cold water on the head. The symptoms were such that the man would have died, but for immediate treatment. It is a remarkable fact that in this case no bottle or vessel could be found in the room, or under the window. The patient hastily summoned his wife one evening, told her that he had taken prussic acid, and immediately fell down senseless on a sofa, without either cry or convulsion, but drawing his breath deeply, forcibly, and slowly. He recovered in about three hours, but had an unusual disposition to sleep, even on the following day. Another remarkable case of recovery from a dose nearly as large occurred to Mr. Bishop. (*Prov. Med. Jour.*, Aug. 13, 1845, p. 517.)

From the facts hitherto observed, we shall not be wrong in assuming that *about twenty grains* of Scheele's acid, at five per cent. (equal to *one grain of anhydrous acid*), or an equivalent portion of another acid, would commonly suffice to destroy the life of an adult. This I believe to be the nearest approach that we can make to the *smallest fatal dose.* In *Reg.* v. *Bull*, tried at Lewes Autumn Assizes, 1860, a question arose respecting the minimum fatal dose of this poison. The accused, a young medical man, was charged with the manslaughter of his mother, a woman æt. 66. He had prescribed for her prussic acid to relieve sickness. He procured for her a bottle of Scheele's acid, said to contain one drachm. He administered four minims to deceased in the morning, and it appeared to benefit her. In the evening he gave her another dose, amounting, according to his statement, to "seven drops." The deceased went upstairs, became insensible, and died in a few minutes. When the bottle was examined twenty-five minims remained in it; hence thirty-five minims were alleged to be missing, but the druggist who sold the acid poured out the quantity conjecturally, and the bottle was found to have a broken cork. The strength of the acid had not been determined. Under these circumstances the prisoner was acquitted. In this case the court desired to know the relation of drops to minims, but no satisfactory answer could be given. The size of a drop materially depends on the nature of the liquid, the mouth of the bottle, and the rapidity of the measurement. Seven drops of Scheele's acid dropped from a small vial measured seven minims. There can be no doubt, in the above case, that the poison caused death, and unless we assume that seven drops or minims will destroy life, which is not probable, the deceased must have taken a much larger dose than the accused had intended.

Period at which Death takes place.—When the dose is two drachms and upwards we may probably take the average period for death at from *two* to *ten* minutes. In Mr. Hicks's case, twenty grains of Scheele's acid destroyed life in twenty minutes. It is only when the dose is just in a fatal proportion, that we find a person survive from half an hour to an hour. In this respect, death by prussic acid is like death by lightning; the person in general either dies speedily or recovers altogether. According to Dr. Lonsdale, death has occurred in man as early as the *second*, and as late as the *forty-fifth* minute. But although death does not commonly ensue until after the lapse of a few minutes, sensibility and muscular power may cease in a few seconds.

While it may be said that those who survive an hour have a good chance of recovery, death may still occur from this poison after the lapse of several hours. In a case which occurred to Dr. Fagge, a man swallowed a drachm and a half of the acid. He became rapidly insensible, but did not die for an hour and a half. (*Guy's Hosp. Rep.*, 1869, p. 259.)

Treatment.—Cold affusion to the head and spine has been found the most efficacious mode of treatment. In a case that occurred to Dr. Banks, a girl took by mistake in medicine, thirty minims of prussic acid. Immediately afterwards she sprang up convulsively from her seat, and fell senseless. Her teeth were firmly set, and her eyes staring and fixed. Stimulants failed to rouse her; the limbs were flaccid; the pupils dilated, and she was wholly insensible; the respiration was slow, and the pulse scarcely perceptible. A stream of cold water from a pitcher was allowed to fall from some height on the region of the spine. In a minute she began to move, and became convulsed; her symptoms abated, and in a few hours she was quite collected. She recovered in a few days; but there is hardly a doubt that she would have died had she not been thus early treated. (*Ed. Med. and Surg. Jour.*, vol. 48, p. 44.) In another case of recovery, cold affusion was successfully resorted to at a later period. (*Med. Gaz.*, vol. 36, p. 104; see also *Prov. Med. Jour.*, March 5, 1845, p. 153, and *Ed. Med. and Surg. Jour.*, vol. 59, p. 72.) The vapor of ammonia may be cautiously applied to the nostrils, and stimulating liniments by friction to the chest and abdomen; but unless the dose is small and the patient is seen early, there can be little hope of benefit from any treatment.

Internal remedies appear to be of no service. The blood is speedily poisoned, and no chemical antidote can reach this liquid to counteract the effects of the poison. If the power of swallowing remains, an emetic may be given, the stomach-pump used, or the throat irritated, to clear the stomach of any residuary poison.

In *Reg.* v. *Belaney* (Cent. Crim. Court, Aug. 1844), a question arose respecting the proper mode of treating cases of poisoning by prussic acid. The prisoner was a surgeon, and he was charged with the murder of his wife, who died in his presence from the effects of a large dose of prussic acid. The medical facts in the case were very simple. There could be no doubt that the poison had been taken, and that it was the cause of death. The nature of the symptoms, their rapid and fatal course, and the detection of the poison in large quantity in the stomach, rendered these conclusions absolutely certain. Again, there was no doubt that the poison had been administered, either intentionally or unintentionally, by the prisoner, *i. e.*, that it was through his act, either criminal or innocent, that the poison was placed within reach of the deceased, and under circumstances which would render it not improbable that she would swallow it by mistake. It was placed in a common drinking-glass in the bedroom in which she was lying, the prisoner being at the time in an adjoining room. He accounted for this strange conduct by saying that he was in the habit of using prussic acid medicinally; that he broke the bottle in trying to remove the stopper; and, in order to save the contents, collected the acid in a tumbler or glass, such as is used for drinking-water! His attention was called off, and he went into an adjoining room, without, as it would appear, making any remark, or cautioning his wife respecting the poison placed in the tumbler, and within her reach!

The presumption of criminality, under such circumstances, had no direct relation to medical evidence; it was a question to be decided by the jury from the facts proved. The medical evidence had, however, two important bearings: 1, the plan of treatment which should be adopted in such an emergency by a medical man; 2, the exact period at which insensibility and loss of consciousness supervene in cases of poisoning by prussic acid. The prisoner, on finding that his wife had swallowed the poison, called for assistance, but did not at the time state the real cause of the symptoms; although it came out in evidence that he must have known that the deceased had swallowed prussic acid. He caused her feet and hands to be put into hot water, and talked of bleeding her; but said it was of no use, as circulation had ceased ("she had no pulse"). He told the first witness, who came to her, that "she would not come to; it was a disease of the heart, and that her mother had died just like it nine months ago." But it was subsequently proved that the prisoner had himself registered the cause of death in the mother as bilious fever! The late Dr. A. T. Thomson, who gave evidence at the trial, was questioned upon the usual remedies in such cases, which he stated to be—cold affusion, ammonia, and stimulants; and very properly expressed an opinion, that what had been done by the prisoner could be of no benefit whatever. The jury acquitted the prisoner. The verdict did not proceed from any defect in the medical evidence; the cause of death was clear, and it was for the jury to determine the value of the moral and circumstantial evidence against the prisoner as the alleged administrator. Of these circumstances, which were exceedingly strong, it is here unnecessary to speak; but the learned judge and the jury, in the opinion of most persons, took a very lenient view of them.

CHAPTER LX.

PRUSSIC ACID—CHEMICAL ANALYSIS—THE SILVER, IRON, AND SULPHUR-TESTS APPLIED TO THE LIQUID AND VAPOR—DETECTION IN ORGANIC LIQUIDS WITHOUT DISTILLATION—PROCESS BY DISTILLATION—DETECTION IN THE TISSUES—IN THE DEAD BODY—CHANGES PRODUCED IN IT BY PUTREFACTION—QUANTITATIVE ANALYSIS.

CHEMICAL ANALYSIS.

PRUSSIC ACID is a limpid colorless liquid. Its specific gravity, when its strength ranges from 2 to 5 per cent. of anhydrous acid, is 0.998. It is, therefore, just barely lighter than water, but it readily mixes with water and alcohol in all proportions. It has a faint acid reaction; if litmus-paper is strongly reddened by it, the presence of sulphuric or of some foreign acid may be suspected. It is sometimes thus acidulated for the purpose of preserving it. Its vapor has a peculiar *odor*, which, when the acid is concentrated, although not at first perceptible, is sufficient to produce giddiness, insensibility, and other alarming symptoms. It was at one time supposed that the *odor* might be present in cases in which it would be impossible to detect the poison by chemical processes, and instances of this are given by Orfila (*Ann. d'Hyg.*, vol. 1, p. 489); by Dr. Lonsdale (*Ed. Med. and. Surg. Jour.*, vol. 51, p. 52), and by Dr. Christison (*Op. cit.*, 1854, pp. 760, 774). Improved methods of research have, however, shown that the acid may be detected in cases in which the odor is not perceptible, as well as in those in which it may be concealed by other odors.

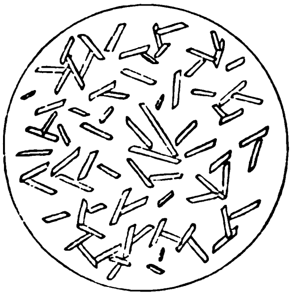
In *Rex* v. *Donellan* (Warwick Lent Assizes, 1781), there was no chemical evidence of the nature of the poison, but the *odor* of bitter almonds was plainly perceived by one witness in the draught administered to the deceased; and this fact, coupled with the symptoms, the rapid death, and the moral circumstances of the case, left no reasonable doubt that prussic acid contained in laurel-water was the cause of death. It was a question in Belaney's case (p. 572), how far the odor of prussic acid was likely to be concealed when the poison was mixed with a saline solution (sulphate of magnesia). From experiment, I have not found that this causes any other difference than mere dilution with an equal quantity of water. Prussic acid is constantly and rapidly evolved from all solids and fluids which contain it, but the evolution appears to be slow in proportion to the degree of dilution.

Its vapor is so diffusible that it readily traverses by osmosis all porous membranes, and, in reference to viscera or organic liquids supposed to contain the poison, unless they are at once securely confined in well-stoppered glass vessels, it will rapidly escape. It traverses the walls of the stomach, and is soon lost. The tests which are best adapted for the detection of this poison, either in liquid or vapor, are equally applicable to the concentrated or diluted acid, so far as the detection of the vapor is concerned, whether it is pure or mixed with organic matter. In the simple state, the tests are three in number,—the *Silver*, the *Iron*, and the *Sulphur* tests.

1. *The Silver Test—Nitrate of Silver.*—This yields, with prussic acid, a dense, white precipitate, speedily subsiding, in heavy clots, to the bottom of the vessel, and leaving the liquid almost clear. The precipitate is identified as cyanide of silver by the following properties: *a.* It is insoluble in cold nitric acid; but when drained of water, and a sufficient quantity of strong acid is added, it is easily dissolved on boiling. *b.* It evolves prussic acid, when digested in hydrochloric acid. *c.* The precipitate, when *well dried*, and heated in a small reduction-tube, yields cyanogen, which may be burnt as it issues, producing a rose-red flame with a blue halo. This is a well-marked character, and at once identifies the acid which yielded the precipitate as prussic acid. By this property, the cyanide is eminently distinguished from all the other salts of silver. In the employment of the silver test for the detection of the *vapor* of the poison, we place a few drops of the silver solution in a watch-glass, and invert it over another watch-glass or beaker containing the suspected poisonous liquid. Cyanide of silver, indicated by the formation of an opaque white film in the solution, is immediately produced, if only in a moderate state of concentration. One drop of a diluted acid containing less than $\frac{1}{50}$th of a grain of the anhydrous acid produces speedily a visible effect. When the prussic

acid is more diluted, a few minutes are required; and the opaque film begins to show itself at the edges of the silver solution. In this case the action may be accelerated by the heat of the hand. If the vapor is allowed to reach the nitrate of silver gradually, and is much diluted with air, then, instead of an opaque film of cyanide of silver, crystals well defined under the microscope will be slowly produced, and these will constitute an additional proof of the presence of the acid in a state of vapor. As shown in the annexed illustration (Fig. 57), these crystals have the form of slender prisms with oblique terminations. They often hang together in groups, and generally require a high magnifying power to render them visible.

FIG. 57.



Crystals of cyanide of silver from the vapor of prussic acid, magnified 124 diameters.

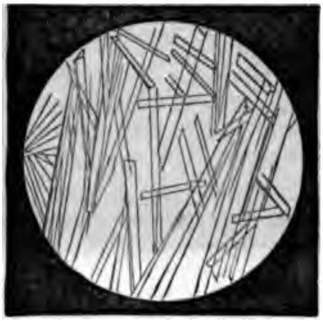
2. *The Iron Test.*—The object of the application of this test is the production of *Prussian blue.* We add to a small quantity of the suspected poisonous liquid a few drops of a solution of potash and of green sulphate of iron. A dirty green or brownish precipitate falls; on shaking this for a few minutes, and then adding diluted hydrochloric or sulphuric acid, the liquid becomes blue; and Prussian blue, of its well-known color, unaffected by diluted acids, subsides. If the prussic acid is in small quantity, the liquid is at first yellow, from the salt of iron formed; it then becomes green, but the precipitate ultimately subsides so as to appear of a deep blue color in the mass. The iron test may be employed for the detection of the *vapor* of prussic acid, by the same method as that described in speaking of the silver test. For this purpose we place a few drops of a solution of potash in a watch-glass or saucer, and invert it over the suspected liquid. After a few minutes' exposure a drop of solution of green sulphate of iron may be added, and then a drop of diluted hydrochloric acid, when Prussian blue will appear. The silver and the iron tests may be easily conjoined in testing the same quantity of poison. If the precipitated cyanide of silver, obtained by the addition of nitrate of silver to the suspected liquid, is dried, and then moistened with strong hydrochloric acid, the vapor may be collected in a watch-glass or saucer, on the plan above described. Prussian blue will be procured, and thus corroborate the action of the silver test. No acid but the prussic will produce Prussian blue under these circumstances.

3. *The Sulphur Test.*—Some years since Liebig proposed the following process for detecting prussic acid as a *liquid.* (*Oesterreichische Med. Wochenschrift,* März 27th, 1847, p. 396.) If a small quantity of the bisulphide of ammonium is added to a few drops of a solution of prussic acid, and the mixture is gently warmed, it becomes colorless, and, on evaporation, leaves crystals of sulphocyanide of ammonium—the sulphocyanic acid being indicated by the intense blood-red color produced on adding to the dry residue a solution of a nearly neutral persalt of iron; this red color immediately disappears on adding a few drops

of a solution of corrosive sublimate. The color is also destroyed by mineral acids, and its intensity is diminished by moderate dilution with water. This process is very delicate, and it therefore requires some care in its application: thus, if the boiling and evaporation are not carried far enough, the persalt of iron will be precipitated black by the undecomposed sulphide; and if the heat be carried too far, the sulphocyanide of ammonium may itself undergo decomposition, and be lost. It will be perceived, too, that it requires a longer time for its application than either the silver or the iron test. If the prussic acid contains traces of Prussian blue or a salt of iron, it will acquire a dark color on the addition of the sulphide.

The great utility of the *sulphur test*, however, is in its application to the detection of the minutest portion of prussic acid when in a state of *vapor*. A watch-glass containing one drop of the sulphide is inverted over the liquid. No change apparently takes place in the sulphide; but if the watch-glass containing it is removed after the lapse of from half a minute to ten minutes, according to the quantity and strength of the prussic acid present, prismatic crystals of sulphocyanide of ammonium will be obtained on gently evaporating the liquid to dryness. With an acid of from three to five per cent. the action is completed in ten seconds. The addition of one drop of the neutral persulphate of iron (free from nitric acid) to the dried residue, brings out the blood-red color instantly, which is intense in proportion to the quantity of sulphocyanide present. Such is the simple method of employing the sulphur vapor test. When the prussic acid is much diluted, the warmth of the hand may serve to expedite the evolution of the vapor. I have elsewhere made some remarks on the application of this process for the detection of prussic acid. (See *Med. Gaz.*, 1847, vol. 39, p. 765.)

FIG. 58.



Crystals of sulphocyanide of ammonium from the vapor of prussic acid (in beer), magnified 70 diameters.

Prussic Acid in Organic Liquids—Detection by Vapor without Distillation.—Any organic liquid suspected to contain prussic acid, *e. g.*, the matters first vomited, or the contents of the stomach after death, may, under the limitations already mentioned, emit an odor of the poison perceptible to one or more individuals. If the liquid has no odor of prussic acid, but an odor of sulphuretted hydrogen, or of some strongly smelling substance, *e. g.*, peppermint or tobacco, still the poison may be present, and it may be detected, if not as a liquid by the ordinary process of distillation, at least by its *vapor*. Of the two processes to be pursued, that which relates to the detection of the *vapor* is the more certain, and open to the fewest objections. It should always be tried before resorting to distillation, because no plausible objection can then be raised on the ground that prussic acid might have been generated from a decomposition of animal matter during this process. If the poison be clearly and unequivocally detected by its *vapor*, there is no necessity for resorting to distillation, except for the purpose of determining the proportion of prussic acid present. The organic liquid should be placed in a wide-mouthed bottle or beaker, to which a watch-glass has been previously fitted as a cover. The capacity of the bottle may be such as to allow the surface of the liquid to be within one or two inches of the concave surface of the watch-glass. The solution of *Nitrate of silver* is then used as a trial-test in the manner already described (p. 474). If the $\frac{1}{200}$th of a grain of prussic acid is present, and not too largely diluted, it will be detected (at a temperature of 60°) by the drop of nitrate of silver being converted into an opaque white or crystalline film of cyanide of silver, the chemical change commencing at the margin. We may then substitute for the nitrate of silver the bisulphide of ammonium, and proceed in the manner above described. The warmth of the hands will facilitate the escape of the vapor, or it may be sometimes necessary to place the bottle in a basin of warm water. If the solution of silver is darkened by sulphuretted hydrogen, as a result of putrefaction, the sulphur test alone should be used.

Detection by Distillation.—This process was originally suggested by Lassaigne. The organic liquid should be distilled in a water-bath at 212°. The apparatus is the same as that for arsenic. (See Fig. 22, p. 320.) About one-sixth or one-eighth of the contents of the retort may be collected in a receiver kept cool by water. The tests may now be applied to the distilled liquid. If the trial-tests indicate that the quantity of poison is small, a solution of nitrate of silver or caustic potash may be placed in the receiver, to fix the acid as it is distilled over; Prussian blue may then be procured in the manner described, or the vapor may be at once absorbed by bisulphide of ammonium in the receiver, and the liquid evaporated to obtain sulphocyanide. Some have advised the employment of sulphuric acid in the distillation process, but this is not necessary unless the liquid is strongly alkaline from ammonia. When the viscera have undergone putrefaction, no trace of prussic acid may be detected either in the form of vapor or by distillation. In this case, it may have been converted into sulphocyanide of ammonium by the sulphide of ammonium produced during putrefaction. This substance may be separated and detected by a process hereafter described. (See p. 582.)

In the Tissues.—Soon after death the poison may be easily detected in the blood, secretions, or any of the soft organs, by placing them in a bottle, and collecting the vapor in the manner already described. This will be found to be far more convenient and satisfactory than the process by distillation. In the case of a dog poisoned by a large dose of prussic acid, Mr. Hicks brought to me the stomach after it had been exposed twenty-four hours, and thoroughly washed under a current of water, and yet the poison was readily detected by placing the whole organ in a bottle, and absorbing the vapor by nitrate of silver. This shows how completely the animal tissues at death are penetrated by prussic acid, and how firmly for a time it is retained by them. The poison has been thus discovered, in experiments on animals, in the blood and in the serous exhalation of the chest.

In reference to the detection of this poison in organic liquids or solids, it may be a question on what results an analyst should rely for conclusive evidence of its presence. In the application of the tests for the vapor, either to an organic liquid or to the product of a double distillation, it appears to me that, in order to render a medical opinion conclusive and satisfactory, the usual chemical results should be at least obtained by the silver and sulphur, or the iron and sulphur tests. So small a quantity of the poison is required for the application of the two tests, that there seems to be no good reason for relying upon the action of one. The silver and iron test may be applied first, and these should be followed by the sulphur test, as the vapor of the latter always contaminates the liquid to be tested. It is true that the sulphur test will reveal the presence of the poison when the iron test fails, owing to the difficulty of collecting a minute trace of Prussian blue; but in this case the quantity of poison must be exceedingly small, and the reaction of the sulphur test very feeble. Although we at present know of no vapor but that of prussic acid which will thus affect the sulphur test, it appears to me that we should not be justified in relying upon infinitesimal results, which admit of no kind of corroboration. The question is here much the same as in reference to the detection by the process of Marsh, of minute traces of what is alleged to be arsenic, when the quantity is too small to be separated by Reinsch's process. The silver test cannot be relied on for detecting small quantities of prussic acid in organic liquids or solids, unless we also procure sulphocyanide of ammonium by the method described. When, however, we have procured the colored results by the iron and sulphur tests, there can be no reasonable doubt of the presence of the poison. With either result separately, as applied to the vapor, there may be room for objecting to the conclusion that prussic acid has been certainly detected. If more than one test is employed by a cautious analyst in examining the liquid poison when in appreciable quantity, how much more is such a corroboration required when he is dealing with an imponderable quantity of vapor obtained from the organic liquids or solids of a dead body?

Period at which the Acid may be detected after Death.—If an organic liquid containing the poison is kept in a closely stoppered vessel, it may sometimes be detected after a long period. But when the liquid has been exposed for three or four days, the vapor-tests have ceased to indicate its presence at the mouth of the bottle containing the liquid; still the acid has been obtained by distillation. I have detected prussic acid when mixed in small quantity with porter after the lapse of twelve months; but then the bottle had been kept closed, and the contents were not putrefied. The practical question for consideration is, for how long a period may we expect to find the acid in the contents of the putrefied stomach or tissues of the body of a person who has died from its effects? The following facts will serve to throw some light upon this question: The acid has been found in the stomach by distillation *seven* days after death, although the odor could not be perceived before distillation. (Case of Ramus, *Ann. d'Hyg.*, 1833, p. 365.) Orfila is said to have discovered it *eight* days after death in the cases of the Parisian epileptics; but he merely states he perceived an odor of bitter almonds, not that he obtained the acid by distillation! In a case in which three drachms had been taken, I could neither detect the acid by the odor nor by the most careful distillation, twelve days after death. The poison has been detected in the stomach by the vapor and by distillation, in one case in three, and in other cases in four, five, and ten days after death. (See Essential Oil of Almonds, *post.*) In the case of Marcooley (Cent. Crim. Court, Dec. 1856), the contents of the stomach consisted of two ounces of a brownish fluid mixed with oil. They were received by the chemical witness *seven days* after death, in a bottle secured with bladder only, at the mouth. Still he detected prussic acid faintly by the smell but distinctly by the tests. He procured from the stomach and contents a quantity of prussic acid equal to six-tenths of a minim of Scheele's strength. In this instance about two drachms of the poison had been taken, and had caused death in an hour.

Assuming that a small but fatal dose has been administered, and that the dead body has been exposed or buried for a few weeks, it is not probable that any of the poison would be found by chemical analysis. The odor may entirely disappear in a week, and the longest period at which the poison itself has been found in the body was in the case of Montgomery (*Report of the Trial of Thompson,* Glasgow Circuit Court, 1857, by Hugh Cowan, pp. 9 and 53). The deceased died in about fifty minutes after having taken two drachms of prussic acid (three and a quarter grains of anhydrous acid). The death took place on the 13th September; the body was buried on the 17th, and exhumed on the 30th. The parts removed were then put into stoppered bottles, and on the 5th of October, the Drs. McKinlay detected prussic acid doubtfully by the odor, but distinctly by the three tests, in the stomach before distillation, as well as in the liquid distilled from the stomach and its contents. They did not succeed in detecting its presence in the tissues. The poison was here detected *seventeen* days after death. About five weeks subsequently to this analysis, the organs, which had been kept closely secured in glass bottles, were examined by Dr. Maclagan. The heart, kidneys, and intestines gave no indication of the presence of the poison, but it was detected by the sulphur test, in the form of vapor, in one-half of the spleen, although there was no odor of the poison. Dr. Maclagan quoted in his evidence a French case, on the authority of a M. Brame, in which prussic acid is said to have been detected in a dead body *twenty-one days* after interment. (Wharton and Stillé, *Med. Jour.*, p. 492.) But the steps of the analysis in this case were so unsatisfactory as to render it doubtful whether prussic acid was discovered at all. It appeared from the evidence in the case of Montgomery that the body was placed under favorable circumstances for the retention of the prussic acid. It is obvious that the answer to this question must in a great measure depend on the dose taken and the time which the person survives.

Another question may here present itself. Is the discovery of prussic acid in the stomach or tissues of a person a proof that death has been caused by it? As a general rule we should be justified in answering this question in the affirmative. We do not here meet with an objection which applies to most other poisons, that the patient may have been cut off by disease supervening after it had been taken; since if this poison operates fatally at all, it is in the course of a few minutes. Latent diseases of the heart and brain may undoubtedly, by a coincidence, cut short life. Prussic acid, it is well known, is used medicinally, and patients are in the habit of taking it for some time after they have ceased to be attended by the medical man who has prescribed it. A person may die suddenly from natural disease while taking the acid, and a chemist, relying on the discovery of the poison in the stomach, might give an opinion that death was caused by it. In such cases, on a recent inspection, the acid would be found in mere traces; if a larger quantity is detected than would correspond to the residue of a medicinal dose, there would be reason to suspect death from accident or suicide; but the whole of the facts of the case should be considered, or the results of a chemical analysis might seriously mislead a jury.

The detection of the acid in the tissues, in the soft organs, or in the blood, would justify the opinion that it had been taken into the body during life. It is a diffusible poison, and will readily traverse the membranous structures; but it must be within the body in order that this should occur. When two or more poisons are present in the stomach, and one is prussic acid in a fatal dose, there can be no reason to hesitate in assigning death to the latter. In a case which occurred in 1837–8, prussic acid and arsenic were found in the stomach after death. In another, the mixture taken by the deceased consisted of brandy, opium, arsenic, and prussic acid.

It has been suggested that the acid might be spontaneously produced after death by chemical changes in the fluid contents of the stomach. Certain articles of food have also been assigned as possible sources of the poison. Bitter almonds and the kernels of stone-fruits, it is well known, contain principles which by contact with water produce an oil and prussic acid. In such a case there would be the odor of the poison, and if the death was recent, a small portion of the acid might be procured by distillation; but a proper examination would show the presence of the seeds producing these traces of prussic acid. Further, symptoms like those of poisoning with prussic acid would not have preceded death. That any mistake should occur, it must be assumed that a person, after eating the seeds, dies suddenly from some natural cause, and the medical expert refers death to the poison, merely because traces of it are discovered in the stomach. Such a condition of things is wholly improbable. The prussic acid may be found, but there may be no seeds to account for its production; the quantity of the acid may be so large as to be utterly inconsistent with this theory; and the symptoms preceding death may or may not have been such as this poison would produce.

In two modern cases this objection to chemical evidence has been raised by medical experts employed for the defence, but in each it was set aside by the facts proved. The first of these is *Reg.* v. *Tawell* (Bucks Lent Assizes, 1845); the prisoner, a Quaker, was indicted for the murder of Sarah Hart. The deceased was found insensible and dying, and no accurate account of the symptoms could be obtained, as no one but the criminal was present. The body was examined eighteen hours after death, but no odor was perceived about the mouth, or in some blood which had been drawn from the body. The lungs were slightly congested, and there were some old pleuritic adhesions, but there was no disease of any organ to account for death. The stomach and bowels presented no morbid change. The contents of the former amounted to twelve ounces of liquid, having no odor of prussic acid, but merely a strongly acid smell of beer. (*Lancet*, April 5, 1845, p. 379; *Northern Journal of Medicine*, May, 1845.) They consisted of partially digested food, intermixed with the pulp of apple, but *without the pips*. Prussic acid was obtained from the contents of the stomach by distillation; it was identified by the application of the usual tests, and after separation as cyanide of silver, by its odor. The quantity thus obtained amounted to *one grain* of anhydrous acid, equal to *fifty minims* of pharmacopœial acid. The administration of the poison to the deceased was clearly brought home to the prisoner, partly by a series of moral circumstances of a most convincing kind, and partly by his own admissions. He unconsciously supplied all that was deficient, *i. e.*, evidence from symptoms preceding death. He attributed death to suicide; but this was entirely out of the question. The learned judge (the late Lord Wensleydale) who tried the case, showed that he was fully competent to unravel and expose the sophistry, legal and medical, brought forward in the defence. In charging the jury, he said: "The statement of the prisoner's counsel that it was a rule of law that there should be *direct proof* of death having been caused by poison, and of the presence in the stomach of a sufficient quantity of poison to produce death, was not true; neither was it necessary to prove what quantity of prussic acid would destroy life by the testimony of a person who had actually seen a human life destroyed by it. With regard to the smell, the only conclusion from the evidence was, that smell was a proof of the presence of the poison, but that the absence of smell was no proof of its absence. According to the witnesses, a grain, or even less than a grain, of prussic acid taken into the stomach was sufficient to cause death. With respect to the allegation that prussic acid might be obtained from apple-pips, Mr. Cooper, the chemical witness, found apple but no pips in the stomach, and it was only by the distillation of the (bruised) pips that the acid was formed." The jury returned a verdict of guilty, and the prisoner, before execution, confessed that he had perpetrated the murder.

It was supposed that the poison in this case was not administered by the mouth, but in a more secret manner, *per vaginam*. This opinion was based on the absence of odor in the stomach, and on a reported confession of the criminal. It was quite inconsistent with the medical facts. One grain of anhydrous prussic acid was certainly found in the stomach, and this dose was of itself sufficient to cause insensibility and rapid death. In reference to the defence here set up, I have found that the seeds of ten common apples distilled with water (the husks of the seeds being unbroken) did not yield in the distillate the slightest trace of prussic acid. When they were bruised and redistilled in a raw state (unboiled), there was a mere trace of prussic acid in the distilled liquid, and a quantity of Prussian blue, equal to the 150th part of a grain was obtained. (See *Med. Gaz.*, vol. 36, p. 328.)

In the case of Montgomery (Glasgow, Dec. 1857), the views of Orfila and other chemists regarding the spontaneous production of prussic acid in a dead body were strongly urged in the defence. Only traces of prussic acid were found in the stomach of the deceased after the long period of seventeen days, and in the preserved spleen nearly two months after death. The late Lord Justice Clerk, in addressing the jury, dismissed the theory as wholly improbable and unsustained by any facts. "If it were true, this acid," he said, "would be found in the body not only in cases of poisoning, but in many other cases. He trusted that it might never be again brought forward in the hope to perplex and mislead a jury, and to try to take off the effect of the clear and decided proof of the existence of poison in the body, and of the possession and use of poison by the accused." (Cowan's *Report*, p. 100.)

Cyanide of potassium may be present in a dead stomach and yield prussic acid by distillation; but this is itself an active poison, and its presence may be easily discovered in the liquid contents before they are distilled. In all cases in which the vapor-tests fail to act, it is advisable to test the liquid before distillation, for the presence of a cyanide or a sulpho- or ferrocyanide.

The discovery of a sulphocyanide in the contents of the stomach or tissues may be of importance. The use of the sulphur test (*ante*, p. 577) shows how readily prussic acid is convertible into sulphocyanide of ammonium in the presence of sulphide of ammonium. As this is generally a result of putrefaction, any of the poison contained in the stomach at the time of death may undergo this conversion. In order to detect it, we dry the viscera and their contents, and digest them in a mixture of one part of alcohol and four of water. The decoction may be filtered and concentrated by evaporation. If much organic matter is present, it may be submitted to dialysis (*ante*, p. 155) and the dialyzed liquid or the solid residue obtained from it, tested by persulphate of iron. A blood-red color, which is discharged by a solution of corrosive sublimate or by boiling the liquid in diluted sulphuric acid, will indicate the presence of a sulphocyanide. The saliva contains traces of a sulphocyanide, but the amount obtained in solution would show whether it was due to saliva or to the converted poison.

I have found that one drachm of Scheele's prussic acid, mixed with part of a stomach, liver, and intestines in a putrescent state, ceased to evolve any vapor sufficient to affect the silver or sulphur tests after the lapse of two days in the month of July. The contents of the jars, loosely covered, were allowed to pass through all the stages of putrefaction, and were examined at intervals of three months and an entire year. On each occasion the presence of a sulphocyanide was clearly detected in quantity by the process above mentioned. In the putrefied body of a person who has died from the effects of prussic acid, we may not find the acid in a free state, but it will be easy to discover this product of its decomposition. If none be found, there would be no evidence that prussic acid was in the body at the time of death.

Quantitative Analysis.—It is sometimes a matter of importance in reference to the fatal dose, the identity of a particular acid, etc., to ascertain the strength of the prussic acid taken or administered. It is much more satisfactory to determine this point by chemical processes than by giving the poison to dogs or rabbits, and noting how long a time it requires for a certain dose to destroy life, or by assuming its strength from its designation. A measured and weighed quantity of the acid may be precipitated entirely by a solution of nitrate of silver. The precipitate should be washed and dried in a water-bath until it no longer loses weight. One hundred grains of dry cyanide of silver are equivalent to 20.14 grains of anhydrous prussic acid; this is in the proportion of about one-fifth, so that the weight of dried cyanide divided by five, gives, with sufficient accuracy for common purposes, the quantity of anhydrous prussic acid present. One hundred grains of the *British pharmacopœial acid* should therefore yield ten grains of cyanide of silver; and the same quantity of *Scheele's acid* from twenty to twenty-five grains. The amount of acid obtained by distillation of the contents of the stomach may be determined by a similar process. Every five grains of dry cyanide of silver represent one grain of anhydrous acid.

CHAPTER LXI.

BITTER ALMONDS AND THE ESSENTIAL OIL—SYMPTOMS—POWER OF LOCOMOTION—APPEARANCES AFTER DEATH—FATAL DOSE—ANALYSIS—ARTIFICIAL ESSENCES—BITTER-ALMOND-WATER—LAUREL-WATER—NOYAU AND OTHER LIQUIDS CONTAINING PRUSSIC ACID.

BITTER ALMONDS AND PEACH-KERNELS—THE ESSENTIAL OIL.

THE bitter almond owes its poisonous properties to prussic acid, which is easily obtained from it in a state of admixture with an essential oil, by distillation with water. It is, however, a remarkable fact that none of the acid exists ready formed in it; and the poison is not produced except through the agency of water on the almond pulp. Heat is not required for this reaction; the mere trituration of the pulp with cold water is sufficient to produce the acid. Several cases are reported by Wibmer, in which serious symptoms occurred in children who had eaten immoderately of bitter almonds. (*Arzneimittel.* Amygdalus.) A girl, æt. 5, was nearly killed by eating a portion of bitter-almond cake. M. Bonjean relates that a cow was poisoned by drinking water into which a small portion of the residue left after the expression of the fixed oil had been put. (*Faits Chimiques, etc.*, p. 56.) There are two instances recorded in which bitter almonds are reported to have caused death in the human subject, but the facts are by no means clearly detailed. Judging from reported cases, a large quantity may be taken, even by children, without necessarily destroying life. Dr. Schlesier met with an instance in which a boy between two and three years of age, ate an ounce of bitter almonds (about 54). A quarter of an hour afterwards there was a general relaxation of the limbs; the countenance was pale, depressed, and drooping; the pupils dilated; respiration sighing; there was also a tendency to sleep, followed by vomiting of the coarsely digested pulp of the almond, which had a very strong smell of prussic acid. Emetics with ammonia, and exposure to a free current of air, soon restored him. (Canstatt's *Jahresbericht*, 1844, B. v. s. 289. See also *Ed. Monthly Journal*, Oct. 1850, p. 379.)

Dr. Samuels, of Wanganui, New Zealand, met with the following case. A boy, æt. 5, ate a quantity of *peach-kernels*, and when seen by Dr. Samuels half an hour afterwards, he was lying in a state of partial coma; the pupils were dilated; the skin was cold and clammy; and the pulse feeble. The first symptoms were dizziness, stupor, fainting, and inability to stand. He vomited an ounce or more of masticated peach-kernels. An emetic and some castor oil were given to him. He slept for three hours, and then recovered. This was a case of poisoning with peach-kernels, in which the poison, prussic acid, was generated by mastication. (*Brit. Med. Jour.*, Sept. 19, 1874, p. 375.)

Essential Oil—Peach-nut Oil.—The essential oil, which is produced by the distillation of the pulp of the bitter almond with water, is a powerful poison, and has caused numerous deaths. In 1837–8, there were only four deaths recorded from poisoning by this oil. In five years (1863–7) there were thirty-one registered deaths from this poison; and it is now a frequent cause of death from accident or suicide. Its taste and odor render it difficult of administration for the purpose of murder; nevertheless, the case of *Reg.* v. *Fisher* (York Lent Ass., 1855), shows that it may be thus used. In this case there was a suspicion that it might have been poured down the deceased's throat while he was asleep.

The poisonous properties of this oil are due to the presence of prussic acid, which is intimately combined with it. Five pounds of the almonds are calculated to yield about half an ounce of the oil, and the quantity of anhydrous hydrocyanic acid contained in it varies from eight to fourteen per cent.—on an average ten per cent. I find, by another calculation, that 2500 parts of bitter almonds yield 100 parts of amygdalin, and these by a reaction with the elements of water, produce 41 parts of essential oil and 6 parts of anhydrous prussic acid: hence 100 grains of bitter almonds (equal to ten in number) would be equivalent to 1.88 grains of essential oil and 0.24 grains of anhydrous prussic acid. One hundred parts of the essential oil would contain 12.76 parts of anhydrous prussic acid, and it would require 833 grains of bitter almonds to represent 100 grains of the prussic acid of the British Pharmacopœia. This oil must, therefore, be regarded in its impure state as a most active poison, being at least four times as strong as the pharmacopœial acid, but it becomes weaker by keeping. Its uncertain strength renders it unfit for internal use; but in France it is given in doses of from one-quarter of a drop to a drop. The oil is sold to the public in quantities of not less than a quarter of an ounce, at the rate of from three to five shillings per ounce. The liquid called

ALMOND FLAVOR, spirit of almonds, or essence of peach-kernels, contains one drachm of the essential oil to seven drachms of spirit. It is sold to the public in quantities of not less than a quarter of an ounce, at the rate of one shilling per ounce, for the purpose of giving a pleasant flavor to pastry, blanc-mange, etc.! It may be as well to state that one ounce of this almond flavor is at the lowest computation equivalent in strength to 250 grains of the pharmacopœial prussic acid. In some cases it may happen to be nearly equal in strength to this poison, and yet it is sold without restriction, and is intrusted in private families in the hands of ignorant cooks to apportion the dose which may give the requisite flavor to food!

Symptoms.—The following may be taken as a summary: sudden insensibility; lividity of the face; eyes glassy, prominent, fixed, and staring; pupils dilated and insensible to light; jaws spasmodically closed; frothy mucus about the mouth; in some cases vomiting of food; coldness of the skin; spasmodic and intermittent breathing, sometimes stertorous; absence of the pulse; head spasmodically drawn backwards, and sometimes the trunk; general relaxation of the limbs; a strong odor of bitter almonds in the breath.

In a case the particulars of which were communicated to me by Dr. Bull, of Hereford, a woman swallowed about seventeen drops of the essential oil, and she died in half an hour. She was seen by Dr. Bull in about fifteen minutes. She was then insensible; her face was livid; the lips were separated; the teeth clenched; there was froth about the mouth; the eyes were half-shut and glassy; the pupils dilated and fixed; there was convulsive breathing with heaving of the chest at intervals; there was no pulse, and the action of the heart was scarcely perceptible. No odor was perceived about the body until after the stomach-pump had been used. The first symptoms observed in this case were strong convulsions, the deceased throwing her arms about as if in pain. There was a short interval between the taking of the poison and the production of insensibility. The deceased called out, and she had had time enough to cork a small bottle which had contained the poison, to put it into a bag, draw the strings of the bag, and hang it over a chair by the side of her bed. A boy, æt. 13, swallowed a quantity of the oil; he was found lying on the floor motionless and insensible; his face pale; his eyes open and fixed, the pupils were dilated, and he was rolling about and panting for breath; the pulse at the wrist was imperceptible; he died in a quarter of an hour without any convulsions appearing. A man, æt. 20, swallowed about two ounces of the oil. A person present saw him fall suddenly while in the act of swallowing, he made a loud cry, gave one deep expiration, and died.

In another case, a woman, æt. 46, who had been in the habit of using the *almond essence* for flavoring confectionery, swallowed about half an ounce (thirty drops of the oil). She died in less than half an hour. When seen by a medical man ten minutes after she had taken the poison, she was perfectly insensible. The face was pale but swollen, and covered with perspiration; the eyes stared fixedly as if in terror; the pupils were dilated. The lips were partly closed and livid, and a frothy mucus issued from the mouth. The lower jaw was firmly contracted, while the muscles of the neck and of the limbs, excepting those of the fingers, were flaccid. She breathed slowly and heavily, making about ten respirations in a minute; the pulse was from 30 to 40, and feeble. There was an odor of bitter almonds in the breath. Some blood which was drawn from the arm was thick and dark, resembling choleraic blood. In spite of the use of the stomach-pump and cold affusion, the patient did not show any signs of recovery, but gradually sank. (*Assoc. Med. Jour.*, Dec. 13, 1856, p. 1055.) In March, 1853, a woman, æt. 39, swallowed half an ounce of *almond flavor*, containing half a drachm of the essential oil. In ten minutes she was seen by Mr. Phillips, of Coventry, who found her perfectly insensible and motionless; the pupils were moderately dilated and insensible to light; the mouth was partly open, the lips were pale, there was no distortion or spasmodic movement of the features; the pulse was slightly tremulous, and entirely ceased in a few minutes; the breathing was slightly stertorous, and took place at long intervals. She continued in this state for twenty minutes without any convulsive movements of the body, when she died, *i. e.*, half an hour after she had taken the poison. In another case two drachms destroyed life in seventeen minutes. (*Lancet*, Oct. 17, 1863, p. 447.)

A case was referred to me for examination by Mr. Savage, in which there was clear evidence of the power of locomotion after probably a large dose of the poison had been taken. The deceased mixed the oil with some ale in a cup, stirred it up with a pipe, and drank off the greater part. Five minutes had elapsed when he was seen deliberately walking towards a staircase apparently conscious and self-possessed, for he replied rationally to a question put to him. The symptoms then came on very suddenly, and commenced with vomiting, during which, probably, part of the oil which he had swallowed was ejected. He became insensible; the breathing was convulsive, and took place at intervals; but, excepting slight opisthotonos, there were no convulsions. From the facts observed by Mr. Savage, it appears probable that the whole duration of this case did not exceed seven minutes; and the fatal symptoms were not manifested until within the last two minutes. In another case this power of locomotion after the taking of the poison was also clearly manifested. A man, æt. 50, swallowed six drachms of the oil of bitter almonds. He then walked down stairs, spoke to his son, and died in about ten minutes. In this case the lungs were remarkably emphysematous, the air-cells being distended into bladders. The heart was full of blood, and the foramen ovale open. (Harveian Society, *Lancet*, Jan. 30, 1858, p. 128.)

Appearances.—In Dr. Bull's case (*supra*), on inspection nine hours after death, no odor of almonds was perceptible in the chest, head, or heart, nor in the venous blood with which the system was gorged. The lungs and heart were healthy. The vessels of the brain were congested, and there was a general effusion of serum on the hemispheres. The mucous membrane of the stomach was much congested. On opening it, the bitter-almond odor was quite perceptible. (See *Prov. Med. Jour.*, Sept. 11, 1844, p. 364.) In the case of the boy (*supra*), which proved fatal in a quarter of an hour, on inspection there was pallor of the face, with lividity of the depending parts; the lungs were congested; the odor of the poison was perceptible only in the abdomen, and very distinctly in the contents of the stomach. The mucous coat of this organ was generally pale, but there were some patches of ecchymosis scattered over it. The essential oil and prussic acid were detected in it. (*Lancet*, July 12, 1845, p. 40.) In a case which proved fatal in three hours, the skin was partially livid, the blood fluid, and the membranes of the brain as well as the lungs were gorged. The contents of the stomach had a strong smell of the oil, and the mucous coat towards the intestinal opening had a red appearance. The other organs were healthy. The blood, with which the venous system is gorged, is generally liquid and of a dark color. In the case of Mr. Sadleir (Feb. 1856), whose death was caused by a very large dose, there was a strong odor of the oil at the mouth, but no froth; the eyes were lifelike and glistening, the pupils dilated. The body was examined forty-eight hours after death. There was congestion of the lungs and bronchial tubes. The right auricle of the heart was distended with blood; the other cavities were empty. The odor of the oil was perceptible throughout the body. The stomach contained ten ounces of undigested food, mixed with from *two to three ounces* of the oil. There was slight congestion of the intestines. The kidneys, as well as the brain and its membranes, were congested. There was an effusion of bloody serum at the base of the brain. In the case of a girl, æt. 18, who died in a few minutes from a dose of almond flavor, equivalent to about thirty drops of the oil (communicated to me by Mr. Hunt, of Bath), the appearances were much the same; there was engorgement of the lungs, with distension of the cavities of both sides of the heart. The blood was fluid. From this it will be perceived that the appearances in the body are by no means uniform. There is commonly a general congestion of the organs with dark liquid blood and an odor of the poison throughout the cavities. In some cases the congestion is most marked in the brain, in others in the lungs and heart, and in others, again, in the viscera of the abdomen.

Fatal Dose.—The smallest quantity of the oil which has yet been known to destroy life was in the case which occurred to Dr. Bull, of Hereford (p. 585). A woman, æt. forty-nine, was in this instance killed in half an hour by a dose of less than twenty drops. Probably not more than *seventeen drops* were taken. A dose of half a drachm, or thirty minims, has destroyed the life of an adult. It may be inferred that a dose of from *twenty* to *forty drops* of the oil containing prussic acid, may prove fatal to adults under ordinary circumstances. Children would die from a still smaller quantity; nevertheless, there is a case on record in which a girl, æt. nine, recovered from a dose equivalent to seven drops.

The largest dose from which there has been recovery was in the following case. A boy, æt. four, swallowed from a bottle, about four or five drachms of the concentrated oil. He replaced the bottle on a table and ran out of the room. He then staggered and fell in a state of insensibility. In five minutes he was seen by a medical man, and he was then laboring under the following symptoms: Countenance flushed, eyeballs prominent and protruding from their sockets with a rolling motion, pupils widely dilated and insensible to light, pulse full and strong but slow, breathing stertorous, complete opisthotonos and frequent convulsive action of the muscles of the face and neck. Cold affusion and the stomach-pump were immediately employed, and the child recovered in about two hours. (*Lancet*, January 13, 1855, p. 34.) The proportion of prussic acid contained in the oil is not stated.

Period of Death.—This poison may destroy life with the rapidity of a strong dose of prussic acid. In one instance, a man fell while in the act of swallowing the oil, and died instantly. In Mr. Savage's case, death took place in *seven minutes* (p. 586). In the greater number of fatal cases, death has occurred within half an hour. A case which fell under the notice of Mr. Wakefield was unusually protracted; the patient survived *three hours*. Like prussic acid, the essential oil either destroys life rapidly, or the person recovers.

Treatment.—The treatment of a case of poisoning by the essential oil of almonds is the same as that directed for prussic acid (p. 572). If the case is seen early, and the patient is not in a state of collapse, the stomach-pump may be used with benefit, the stomach washed out and the use of this instrument continued until the liquid withdrawn has lost the odor of bitter almonds.

Analysis.—The *essential oil*, which is often called peach-nut oil, is colorless when pure, but it commonly has a pale yellow color, and a strong odor of bitter almonds, by which it may be at once identified. It has a hot, burning taste, and a feebly acid reaction. It produces, when dropped on paper, a greasy stain, which does not entirely disappear by the application of heat. It has a sp. gr. of 1.043; it sinks in water, which dissolves about one-thirtieth part. It is soluble in alcohol and ether in all proportions. When mixed with a few drops of strong sulphuric acid, it forms a rich crimson-red liquid, which, if exposed to air, acquires a yellow color. When poured into cold water, the crimson liquid is immediately destroyed, and a yellow coloring matter falls in globules. The smell and taste of this oil are sufficient for its identification; but nitrobenzole possesses a similar color and odor, and has been mistaken for it. When pure and free from prussic acid, the essential oil is rapidly converted by oxidation into an innocent substance, crystallized benzoic acid. The impure oil undergoes this change very slowly and only partially.

The vapor of prussic acid does not so readily escape from this oil as from the watery solution: hence the vapor-tests do not give the same characteristic results. *Tests.* 1. Add to one or two drops of the oil a like quantity of bisulphide of ammonium. Mere mixture at a low temperature only produces sulphocyanide after standing ten minutes or longer; but if the liquid is warmed to 100°, the conversion is immediate, and the change is indicated by the blood-red color struck on adding to the liquid persulphate of iron. If any unchanged sulphide should give a black color, this may be removed by the addition of one or two drops of hydrochloric acid. 2. Dissolve one or two drops of the oil in alcohol and add to the mixture a few drops of a solution of potash, followed by a solution of green sulphate of iron and hydrochloric acid (p. 575). Prussian blue is formed on agitating the mixture, but it does not appear until the precipitated oxide of iron is dissolved by the addition of diluted sulphuric or hydrochloric acid. The silver test is inapplicable to the oil in its ordinary state. The vapor of the oil produces no change in a drop of a solution of nitrate of silver inverted over it, except after long exposure. If, however, the oil is warmed and it contains prussic acid, there is an immediate production of cyanide of silver. The two tests above mentioned, combined with the odor, are sufficient for all practical purposes.

Organic Liquids.—The odor of the oil would in general indicate its presence in any organic liquid. Owing to its great density, the oil may be found at the bottom of the liquid, while the prussic acid may be partly dissolved in the watery portion. The liquid may be distilled in the usual manner, and the oil and acid in the distillate examined by the tests above described. As ether readily dissolves the oil, this may be in some cases used as a medium for separating it from these liquids. The oil has not been found in or separated from the tissues, but it appears to undergo oxidation in the system. It is eliminated in the urine in the form of hippuric acid. In administering the oil deprived of prussic acid to rabbits, Dr. Maclagan found hippuric acid in the urine. (*Pharm. Journal*, December, 1853, p. 278.)

The pure Oil deprived of Prussic Acid.—This is known to chemists under the name of hydruret of benzyle. When deprived of prussic acid the energy of the oil as an active poison ceases; but it still retains a noxious action on the animal system. It now requires to be given in large doses, and its mode of operation is different. (*Pharm. Journal*, July, 1847, p. 11.) Dr. Maclagan found that a few drops of the oil deprived of prussic acid did not act as a poison on animals; but in larger doses, *i. e.*, a drachm and upwards, it was fatal to rabbits. Two drachms of the pure oil caused a rabbit to fall on its side in ten minutes, and it died in fifty minutes. (*Pharm. Journal*, December, 1853, p. 278.) Some experiments on the oil freed from prussic acid by Mr. Langdale are reported in the *Lancet*, January 10, 1857, p. 45. One drachm given to a middle-sized dog, half a drachm to a cat, and four drops to a rat, did not destroy life, while four drops of the common oil destroyed a rat in two instances. Mr. Price Jones gave fifteen drops of the purified oil to a rabbit. The animal uttered a few cries, but recovered in about ten minutes. Thirty drops given to another rabbit produced violent convulsions, with prostration and oppressed breathing. The animal died in a minute and a half. Five drops of the ordinary essential oil killed a rabbit in about a minute. These facts, viewed together, show that the purified oil possesses noxious properties, although in a much smaller degree, than the ordinary oil. Mitscherlich states that he found it still poisonous when quite free from prussic acid (*Pharm. Journal*, vol. 10, p. 83); but it is a matter of great difficulty to deprive it entirely of the acid. For testing the purified oil, we may employ sulphate of iron and hydrochloric acid, as in the use of the iron test for prussic acid. The production or non-production of Prussian blue will show the presence or absence of prussic acid. The sulphur test directly applied to the oil will equally reveal the presence of the poison.

At an inquest in a case of death from almond flavor, a druggist who sold the poison stated that he purchased it free from prussic acid, but that it generated the poison by age! It need hardly be said that this statement is erroneous. The acid exists in the oil *ab initio* as a result of distillation. There is no after-production of acid, but rather a slow removal of it by oxidation and by its volatility.

In the use of poisonous compounds for flavoring food, it is usually considered that the small quantity required for this purpose cannot be productive of mischief; but it is forgotten that the liquid is employed by ignorant cooks, who apportion the dose of poison by conjecture, and assuming that the greatest precautions are taken, it appears to me that a flavor is at all times dearly purchased if it depends on even a small dose of poison. Mr. Streeter met with a case in which a child suffered from symptoms of poisoning owing to its having eaten tapioca flavored with the oil of almonds. (*Med. Times and Gaz.*, December 16, 1854, p. 625.) There is a liquid sold for flavoring confectionery, under the name of *Essence of Jargonelle Pear.* It is a noxious artificial compound, made by distilling oil of grain or fusel oil with acetate of potash and sulphuric acid. In the *Pharmaceutical Journal* (November, 1851, p. 214), it is stated that a child who had on two occasions eaten confectionery flavored with essence of pear, became partially comatose, with livid lips and a feeble pulse. Symptoms resembling those of poisoning, occasionally observed among children, may often be referred to the eating of confectionery, colored or flavored with various kinds of poison. Among the artificial fruit-essences is one named *Essence of Ribstone Pippin*, or "oil of apples." It is procured from a mixture of bichromate of potash, sulphuric acid, and amylic alcohol. (*Chemical Record*, January 17, 1852, p. 44); all substances of a noxious nature. Butyric ether dissolved in six parts of alcohol is used as *Essence of Pineapple.* This ether appears to constitute the flavor of the pineapple, melon, and strawberry. Impure glycerin (the sweet principle of soap) mixed with alcohol, produces, by fermentation, a similar essence.

BITTER-ALMOND-WATER.

This water is made by distilling one part of almond-cake with eight parts of water. It varies considerably in strength. The late Dr. Gregory has stated that it contains one per cent. of anhydrous prussic acid. The late Mr. Jacob Bell informed me that in a specimen which he analyzed, the proportion of acid was only 0.27 per cent. I have met with specimens containing less than this. The odor is no criterion of the strength, since the odor of prussic acid is concealed by that of the bitter almond, and the odor may exist in a specimen of the water which contains no prussic acid. Its strength is impaired by keeping; thus Zeller found that one ounce of the water fresh made, yielded 5.12 grains of cyanide of silver; but after one year, when kept merely corked in a bottle, the proportion yielded was only 4.62 grains. (*Pharmaceutical Journal*, Feb. 1846, p. 371.)

Symptoms and Effects.—This water is poisonous, and on one occasion the improper use of it led to a criminal trial. (*Reg.* v. *Cronin*, Central Criminal Court, April, 1847.) The accused was charged with the manslaughter of a woman under the following circumstances. He had been in the habit of using a preparation which he called bitter-almond-water, made by mixing three drops of the essential oil with a pint of water; a harmless mixture in small doses. He wrote a prescription for the deceased, in which occurred the words *aquæ amygd. amar.* Six ounces of this were ordered, and the mixture contained besides a small dose of prussic acid. The chemist who prepared the mixture put into it six ounces of the liquid commonly known as "bitter-almond-water" (distilled from the cake). The deceased took a tablespoonful and a half. In three minutes she said: "Oh, how queer I feel!" She left the room, and ran out towards the garden, where she fell, breathing hard and groaning. There was dilatation of the pupils, with general relaxation of the limbs; but there were no convulsions. She died shortly afterwards. There was no doubt that the bitter-almond-water had caused her death. The viscera were generally healthy. There was no odor in the abdomen; but it was perceptible in the brain, the vessels of which were somewhat congested. Prussic acid was detected in the stomach. The accused was acquitted, as it was not considered that he was strictly responsible for the result. (*Med. Gaz.*, vol. 39, pp. 388 and 695.) The quantity of anhydrous prussic acid which the deceased took was equivalent to 0.94 grain; thus bearing out, in a most striking degree, the assumed fatal dose of this poison (p. 570), and proving that dilution with water does not prevent or materially retard its action on the body.

This water is not commonly employed medicinally in England, as its effects, like those of the oil, are very uncertain. Eighteen drops have been known to produce giddiness, dimness of sight, and a tendency to sleep. Twenty-two drops caused convulsions and vomiting. MM. Duvignan and Parent, who tried these experiments on themselves, did not feel inclined to carry the dose further. A drachm of the water killed a moderate-sized dog. (*Paris Med. Jur.*, p. 243.) In France, bitter-almond-water is used medicinally, in doses of from ten to forty drops.

Analysis.—The water is sometimes opaque, from a little oily matter diffused through it; but it may be rendered clear by alcohol. Some specimens will yield readily all the usual reactions with the liquid tests for prussic acid; but when the water has been long kept, and is much diluted, it has often only the odor of the bitter almond, and contains no detectable quantity of prussic acid. When it has been sufficiently strong to cause serious symptoms or death, there will be no difficulty in detecting prussic acid by the vapor or liquid tests. A few drops of the water warmed with the bisulphide of ammonium will give, on the addition of a persalt of iron, the red color indicative of sulphocyanic acid (p. 575). The strength of bitter-almond-water is by no means proportionate to the quantity of bitter almonds used; but it varies according to the process employed for its production. The same weight of almonds has given two kinds of water; one ounce of one yielding as much as 5.35 grains of cyanide of silver, and one ounce of the other only 2.5. When the residuary almond-cake has been previously digested in spirit, the water obtained is always weaker. (*Pharm. Jour.*, Feb. 1846, p. 371.) In this case the amygdalin is partly removed from it.

LAUREL-WATER—LAUREL OIL.

Laurel-water is a weak solution of prussic acid, containing only about one-fourth of a grain per cent. of the strong acid; but it is said to be more poisonous than this quantity of acid would indicate. (*Pereira*, vol. 2, pt. 2, p. 279.) Laurel leaves gathered in wet and cold weather are said to yield more prussic acid than those gathered in hot and dry weather. (*Zeller.*) The old leaves yield much less oil and acid than the young and unexpanded leaves. In some specimens of the water which I procured by distilling the bruised tops and young shoots of the laurel with water, the odor was powerful; but the proportion of prussic acid present was smaller than that above stated. Like bitter-almond-water, it is variable in strength. Specimens long kept and frequently exposed seldom contain any prussic acid, although the odor of bitter almonds may be strong. It is a limpid, colorless liquid, producing, in large doses, the usual effects of poisoning by prussic acid.

By distillation with water the leaves of the plant yield also an essential oil, *cherry-laurel oil*, resembling that of the bitter almond, but much weaker, as it contains on an average less than three per cent. of prussic acid. A fluid ounce of water will dissolve only 3.25 grains of the oil. Every part of the plant is poisonous, but especially the leaves, flowers, and kernels of the fruit; these, when bruised with water, yield prussic acid, but the pulp of the cherry is not poisonous. Articles of food are often flavored with the leaves, and accidents are said to have arisen from this practice. (*Pharm. Jour.*, July, 1847, p. 13.) The late Dr. Paris states that several children were severely affected by partaking of some custard flavored with laurel leaves, and were ill for three days. A girl of six and a boy of five years of age, fell into a profound sleep, out of which they could not be aroused for ten hours. (*Med. Jour.*, vol. 2, p. 402.) The leaves are often employed with impunity; but the proportions of oil and prussic acid are liable to vary with the age of the leaf. Dr. Christison states that he has found ten times as much oil in the young as in the old leaves, when both were gathered in May and June. (*Op. cit.*, p. 788.)

Symptoms and Effects.—About half a teaspoonful of a mixture, consisting of four-fifths cherry-laurel-water, was given by mistake to an infant eight months old. The child drew its head back, was convulsed, and died in a few seconds. The laurel-water taken in this case, is said to have been stronger than usual. The body was inspected twenty-four hours after death. Nothing was observed in the brain and spinal marrow; but the stomach contained two teaspoonfuls of a yellowish liquid without odor, and its mucous membrane was reddened towards the greater curvature. No trace of prussic acid was found in the contents; but the poison was easily detected in the liquid remaining in the vial. (*Med. Gaz.*, Jan. 1843.)

The following case is remarkable chiefly from the circumstance of the symptoms coming on slowly. A man swallowed one morning an ounce and a half of laurel-water. No symptoms appeared until *three hours* afterwards. There was then numbness of the hands and feet, drooping of the head, and involuntary evacuations. The limbs were cold, and he had lost all power over them, although sensibility was retained. The pulse was small; there was perfect consciousness. He gradually became weaker, and died the same evening. On inspection, the only remarkable appearance was, that the blood was viscid and of a dark color. There was no odor of bitter almonds. (Canstatt, *Jahresbericht*, 1844, vol. 5, p. 289.) A man, æt. 60, swallowed two ounces of laurel-water of the Prussian Pharmacopœia. He fell almost immediately, as if in a fainting fit. He vomited some food which he had taken shortly before. He was seen in an hour by Caspar, who found him on a sofa in a half-sitting posture, with his head falling forwards. His face was pale and cold, and the skin generally was cold. The pulse was slow, soft, and irregular. The most striking symptom was a general paralysis of the nerves of motion. He gave no sign of consciousness. The features were occasionally distorted with convulsive movements. There was no power of swallowing; and in this state the man continued, in spite of treatment, for *five hours*, when he died—probably, the longest duration of any case of poisoning by prussic acid yet recorded. The only appearances in the body were congestion of the brain and the right side of the heart, with a dark and liquid state of the blood. There was a smell of bitter almonds throughout the body. (Casper, *Ger. Med.*, vol. 1, p. 431.) The appearances are similar to those met with in death from diluted prussic acid. Similar *treatment* is required (p. 572.)

Cases of poisoning with laurel-water are not common, and generally arise from accident. It has, however, been used for the purposes of murder, and of this the trial of Captain Donellan, at the Warwick Lent Assizes, in 1781, is a remarkable illustration. The accused was charged with the murder of his brother-in-law, Sir T. Boughton, by causing to be administered to him two ounces of laurel-water, which he had criminally substituted for an innocent purgative draught. Admitting that the laurel-water had no greater strength than that above assigned, the deceased must have taken at least *two* grains of pure hydrocyanic acid, a quantity equal to *fifty drops* of Scheele's prussic acid. The draught was administered to the deceased by his mother, Lady Boughton. She perceived at the time that it smelt strongly of *bitter almonds*, the only evidence of the probable nature of the poison; for the original draught, containing rhubarb, jalap, spirits of lavender, and nutmeg-water, would have had no such smell. The following were the symptoms: "In about two minutes after swallowing the draught, the deceased appeared to struggle very much, as if to keep it down, and had a rattling and gurgling at his stomach. In about ten minutes, he seemed inclined to doze; and in about five minutes afterwards, he was found with his eyes fixed upwards, his teeth clenched, and froth running out of his mouth." He died in half an hour after swallowing the draught. An examination of the body was not made until eleven days after death. The appearances were ambiguous, and no poison was detected in the body.

In making every allowance for such coincidences, in the supervention of fatal disease at the time of taking medicine or food, elsewhere pointed out (p. 85), I do not think there is any reason to doubt that in this case the deceased died from poison, and that the prisoner was properly convicted. The medical evidence was strong, whether we regard the time of the occurrence of symptoms, their character, their rapid course, or the period within which death took place. To exclude all notion of these effects depending on a draught just before taken, and having the decided odor of a liquid known to be capable of producing such symptoms, an odor which the originally prescribed draught could not possibly have had, and to refer them to a disease (epilepsy), unusual in so young a subject, and unlikely to have caused death so rapidly or under the symptoms actually witnessed, is simply to create impunity for the cunning and skill often displayed in murder by poison. The insisting upon *direct* evidence only, in such cases, is tantamount to asking for impunity for the educated and skilful or the professional poisoner, and inflicting the full penalty of the law only on the uneducated and unskilful assassin.

Analysis.—The odor of the water is sufficient to identify it, but this will not prove that it contains prussic acid. In order to obtain this proof, it must be submitted to analysis. The following is the result of an examination of a very weak specimen: Nitrate of silver produced no perceptible effect with one drachm of it when the liquids were mixed, nor could any Prussian blue be procured from a like quantity by the use of the iron test. One drop of bisulphide of ammonium added to three drops of the water and heated, gave the clearest evidence of prussic acid by the production of the red sulphocyanate of iron, when the persulphate of iron was added to the evaporated residue. From five to ten drops placed in a watch-glass, produced no film (by the vapor) on nitrate of silver after the lapse of twenty minutes; in the same period of time, one drop of bisulphide of ammonium absorbed the vapor, and left, on evaporation, a perceptible quantity of sulphocyanide. Prussic acid is easily detected, by the sulphur vapor test, in the shoot of the laurel, or in the seed of the berry (not in the pulp) when bruised and mixed with *cold* water. The application of heat to the pulp expedites the chemical change. The bisulphide of ammonium, added to the watery liquid of the pulp and heated, produces the usual reaction of sulphocyanide of ammonium with a persalt of iron.

As it is desirable to have corroboration of the action of the suphur test when the analyst is dealing with small quantities, the following process will be found to present a satisfactory method of employing the silver and sulphur tests with one and the same portion of vapor. Receive the vapor on a drop of solution of nitrate of silver, as described at p. 587. Examine it for crystals by the microscope; if the white film is slowly formed, these will be visible. Now add a drop of bisulphide of ammonium and warm the liquid; black sulphide of silver will be separated, and by slow evaporation a watery-looking residue will result. This may be tested by adding persulphate of iron to the liquid; or clean bibulous paper dipped into it will remove the clear liquid from the black sulphuret of silver, and when the paper is touched with a solution of colorless persulphate of iron, the red color of the sulphocyanide of iron will appear. The strength of laurel-water is so variable that it admits of no safe comparison with prussic acid; each specimen will require a separate examination. An *aqua lauro cerasi* is used in the British Pharmacopœia. It is prepared by distilling one pound of the fresh leaves of common laurel crushed and macerated with two pints and a half of water. One pint is obtained by distillation. The dose is from five to thirty minims. The French Codex prescribes the dose of from ten to forty drops every two hours. (*Pharm. Jour.*, February, 1846, p. 372.)

The distilled water of the leaves of the *Acacia* and of the fruit of the Passion flower contain prussic acid. The acacia-water has a strong smell of bitter almonds, and eight ounces of it, precipitated by nitrate of silver, yielded 4.15 grains of cyanide. The dried leaves gave no prussic acid on distillation. The leaves of the peach yield a water as strong as that of the laurel. The distilled water of the *leaves* of the *sweet almond* also contains prussic acid. Zeller found that one ounce gave 0.575 grains of cyanide of silver. The flower of the common lilac is said to yield traces of this poison.

NOYAU—CHERRY RATAFIA—APPLE-SEEDS—PEACH-KERNELS.

These, and all liqueurs having the smell of bitter almonds, are considered to be poisonous when taken in large doses. The quantity of prussic acid present in them is liable to vary; it may be separated by distillation at a gentle heat, and then tested. I have found that an ounce and a half of good noyau, having a strong odor and flavor, yielded when distilled to two-thirds, scarcely a trace of prussic acid either by the silver or iron test. It had been kept some years in a well-corked bottle. An equal quantity of cherry ratafia, similarly tested, gave no ponderable quantity of Prussian blue. There are other plants, the leaves and kernels of which yield prussic acid; these are, the Bird-cherry, the Peach, Nectarine, Damson, Mountain-ash, Apricot, and the seeds of apples and pears. A case is reported in the *Journal de Chimie Medicale*, 1853, p. 38, in which a child, æt. two, suffered severely in consequence of having eaten ten or twelve kernels of the apricot. I have examined the seeds of oranges and figs, but have found none; nor could I obtain the slightest trace of prussic acid from the distillation of three hundred grains of the *sweet* almond. The quantity produced from the seeds of apples has been grossly exaggerated (p. 581). The seeds of two large apples (seventeen in number) were equivalent in weight to one bitter almond (ten grains); but in the apple-seeds, the quantity of husk is so large that it would probably require the seeds of four apples to produce as much prussic acid as a bitter almond. Ten bitter almonds, weighing 100 grains, will yield by distillation 0.24 grains of anhydrous prussic acid. It would require the seeds of forty apples to produce this quantity. In the unbroken state they would yield none, and they would pass through the body unchanged. If boiled (as in cooked apples), so that the albuminous portion is coagulated, they would yield none, whether broken or unbroken. I have elsewhere alluded to the assumed production of a fatal dose of this poison from one of the most common articles of food (Case of Tawell, *ante*, p. 581). To have accounted for the quantity of prussic acid found in the stomach of the deceased in this case, it must have been assumed that the woman had eaten either 160 apples, or the pips removed from them. Although this theory to account for the presence of poison was supported by the pathetic eloquence of an eminent counsel, the jury declined to accept it. Apple-seeds are not so poisonous as this theory would imply.

Fresh and dried cherries, as well as the kernels and stones, yield prussic acid by distillation with water.. The quantity yielded by the pulp of the cherry is very small, amounting to mere traces, but it is much greater in the stones and kernels. From sixteen ounces of cherry-stone-water, Geiseler obtained 1.9 grains of cyanide of silver; and from cherry-kernel-water, the kernels being to the water as 1 : 8 by weight, the cyanide of silver obtained from sixteen ounces, was equal to 2.36 grains. Twelve ounces of the *kernels* yield 7 grains of hydrocyanic acid; but the proportion of prussic acid yielded by the same weight of cherry-*stones*, according to Geiseler, was not more than 2.3 grains. (*Pharm. Jour.*, Feb. 1846, p. 275.) These kernels, or bitter almonds bruised, are much employed for the purpose of giving a nutty flavor to alcoholic liquids (British sherry and port). It is not often that they are used in such quantity as to occasion accidents; but the following case, the details of which are imperfectly given, will show that the eating of a large quantity of the kernels may operate fatally.

A girl, æt. 5 years, ate a large quantity of the kernels of sweet cherries (*Prunus avium*). Her brother (a few years older than herself) also ate some. After the lapse of a few hours symptoms of poisoning appeared. When seen by a medical man on the next day, the girl was in such a state of stupor that she could not be roused. The eyes were closed, pupils considerably dilated, the skin moist and hot, respiration exceedingly hurried, pulse small and quick, and the evacuations were discharged involuntarily; the child was very restless. An effervescing mixture was ordered internally, and cold fomentations were applied to the head; after a few hours, vomiting of a greenish substance ensued, and this was followed by retching, which continued until death; the body was spasmodically drawn backwards (opisthotonos). The illness lasted forty hours. On an examination, the stomach was found intensely reddened; the intestines were strictured and invaginated (intussusception), but there was not any inflammation. The liver, spleen, and large vessels contained a black tar-like blood. The boy, who had eaten fewer cherry-kernels, was likewise ill, but recovered in the course of a month. An eruption analogous to nettlerash showed itself on the arms of both children; they were both perfectly well (according to the statement of the mother) before eating the cherry-kernels, and no other cause for the attack could be assigned. The kernel of the Prunus avium (Cerasus nigra) contains amygdalin, and produces prussic acid as well as essential oil in the stomach. (*Philadelphia Med. Exam.*, July, 1845, p. 490.)

A singular case of poisoning by *peach-kernels* was communicated to me by Mr. Hicks. A medical man swallowed half an ounce of liquid made by digesting gin on a large quantity of peach-kernels. He became giddy, and had violent constriction of the throat and dimness of sight. He vomited and recovered. The bottle was brought to me by Mr. Hicks. A few drops of the liquid contained in it yielded only a faint trace of Prussian blue. The kernels weighed 124 grains; they were large, and the skins entire. All the amygdalin must have been extracted, for on bruising the kernels with water and distilling them, not a particle of the poison could be procured. The amygdalin may have been dissolved by the spirit and converted into prussic acid in the body.

JATROPHA MANIHOT—CASSAVA.

The root of one variety of this West Indian plant, known under the name of Bitter Cassava, contains in its juice prussic acid. It is, therefore, when recently expressed, highly poisonous, inducing coma, convulsions, and death. Prussian blue may be obtained from the fresh juice by the iron test for prussic acid. The vegetable principles of the plant, evaporated to dryness, form what is called *Cassava-cake*, which is not only inert, by reason of the poison being volatilized, but highly nutritious. The starch obtained from this root is well known under the name of *Tapioca*. Neither cassava nor tapioca yields any trace of prussic acid.

CHAPTER LXII.

CYANIDE OF POTASSIUM—SYMPTOMS AND APPEARANCES—LOCAL ACTION—FATAL DOSE—ANALYSIS—SULPHO- AND FERROCYANIDE OF POTASSIUM—CYANIDE OF IRON—PRUSSIAN BLUE—CYANIDES OF MERCURY AND SILVER.

CYANIDE OF POTASSIUM.

PRUSSIC ACID is as fatal to animal life when its elements are combined with alkaline bases as when it is free. Thus it has been found that the same quantity of diluted prussic acid will kill a dog, whether it be given in a pure state or combined with ammonia or potash. (*Orfila*, vol. 2, p. 292.) Hence, ammonia cannot be regarded as a chemical antidote in cases of poisoning by prussic acid, but merely as a stimulant. Alkalies have not even the power of fixing the acid. In *Reg.* v. *Cronin* (C. C. C., 1847), the prisoner had prescribed aromatic spirits of ammonia with prussic acid, and the question was, whether the poison would become thereby in any degree disarmed of its virulence. The answer was in the negative, as the rapid death of the woman proved. When mixed with an alkaline base, such as ammonia, prussic acid is liable to undergo speedy decomposition. It becomes yellow, then brown, and finally almost black, a thick black sediment being formed in it. This change is, however, only partial; in one specimen thus altered, I found a large quantity of free prussic acid after two years. The change is not observed to take place when the prussic acid bears a small proportion to the base, or *vice versâ;* nor does it so readily occur when the prussic acid is much diluted.

The only saline compound of prussic acid which is of any practical interest as a poison is the Cyanide of Potassium.

Symptoms.—This salt has a bitter taste, producing first a sense of coldness on the tongue, followed by a feeling of constriction and burning heat in the throat. It is one of the most formidable poisons known to chemists. It has destroyed life in a quarter of an hour. A dose of five grains has proved fatal in three instances. In one case the person died in two hours. (*Chem. News,* Sept. 5th, 1863.) The symptoms which the cyanide produces are similar to those occasioned by prussic acid—insensibility, spasmodic breathing, convulsions, with tetanic stiffness of the jaws and body. They appear in a few seconds or minutes, and run through their course with great rapidity.

Insensibility is not always an immediate symptom. A woman, who was at the time under medical treatment, took by mistake a teaspoonful of a solution of cyanide of potassium, containing about seven grains of the salt. Immediately after taking it she complained of a severe burning pain in the stomach, and a feeling as if the bowels were about to act. She went to the water-closet, and her strength left her. She was removed to bed, and speedily became unconscious. It was found impossible to introduce anything into the stomach. She died in less than an hour. There was no convulsion before death, but a sudden convulsive action of the body took place after the heart had ceased to beat. The appearance of the body was so natural, even on the day following death, that some of her friends supposed there might still be life. (*Boston Medical and Surgical Journal,* Dec. 11, 1856, and *Brit. and For. Med. Rev.,* 1857, vol. 19, p. 528.) In June, 1856, a woman swallowed an ounce and a half of a solution of cyanide of potassium, used for photographic purposes. The quantity taken amounted to five grains. In two minutes she became unconscious, the whole of the body was slightly convulsed, and the pupils of the eyes were dilated. She foamed at the mouth, the pulse was small and feeble, and there was spasmodic closure of the jaws. Nevertheless, as she had lost some teeth, there was sufficient space for the introduction of the tube of the stomach-pump within five minutes after she had taken the poison. She died in twenty minutes. In the Registrar-General's Report for Oct. 3, 1857, three deaths are stated to have occurred from this salt among the families of soldiers; two were cases of suicide, and one of accident. It appears that they employed it for cleaning lace. In the five years, 1863–7, one hundred and fifty-one deaths are reported to have been caused by prussic acid and cyanide of potassium. (*Reg.-Gen. Rep.*) (For cases of its fatal action see *Med. Times and Gaz.,* Oct. 12, 1850, p. 390; Nov. 9, 1850, p. 482; and July 12, 1851, p. 41; also *Chem. News,* April 27, 1861, p. 260.) The salt is much used by photographers, and has given rise to many accidents among persons engaged in the practice of this art.

Appearances.—In a case in which an inspection of the body was made two days after death, there was no remarkable odor; the muscles were stiff and rigid; the face and forepart of the body pale; the back part livid, except those portions which had sustained pressure. The fingers and toes were convulsively bent inwards, the nails blue, eyelids half-closed, lips pale, the vessels of the brain filled with bluish-red (blaurothem) blood. On making a section of the brain and spinal marrow, bloody points were observed. The lungs were congested posteriorly, and on cutting into them a strong odor of bitter almonds was perceived. A yellowish mucus was found in the stomach, which yielded on analysis cyanide of potassium. The mucous membrane was reddened near the intestinal end. The poison was not detected in any part of the body except the contents of the stomach and intestines. (Casper's *Wochenschrift*, Oct. 4, 1845, p. 657.) In November, 1851, a girl, æt. 18, was brought to Guy's Hospital. Half an hour before her admission, she was seen to swallow a solution of cyanide of potassium. She vomited once. It is stated that she was alive when put into the cab, but when taken out at the hospital, seven minutes afterwards, she was completely insensible, pulseless at the wrist, and to all appearance dead. Artificial respiration was at once resorted to, ammonia was applied to the nostrils, warmth to the limbs, and cold affusion to the spine; but all to no effect, and, with the exception of an alteration in the pupils observed during the first few minutes of her admission, she evinced no signs of vitality. The body was inspected on the following day. The stomach contained a large quantity of half-digested food; its mucous membrane was of a pink color, and deeply injected, especially in patches. Every other organ was healthy, and there was no appearance of corrosion about the mouth. The contents of the stomach were of a pale straw-color, semifluid, and had a decidedly bitter almond odor. Prussic acid was detected in them. (See also a case by Dr. Ellis, *Lancet*, Oct. 17, 1863, p. 447.)

This poison is generally fatal. Two cases of recovery from large doses are, however, reported by Mr. Stevenson. (*Lancet*, 1871, vol. 1, p. 806.) A rare instance of recovery was communicated to me by Mr. Taafe, of Brighton. In March, 1862, a man swallowed the greater part of a solution containing an ounce of the commercial cyanide, which he had dissolved for the purpose. Mr. Taafe found the man a few minutes afterwards lying in the street insensible, and breathing stertorously; and in about ten minutes he applied the stomach-pump, with cold affusion, freely. In two hours the man vomited, and from that time rapidly recovered. The commercial cyanide frequently contains a large proportion of carbonate of potash. This lowers its poisonous action. The cyanide may be separated from the carbonate by its solubility in rectified spirit.

Cyanide of potassium has a local chemical action upon the skin; and if this is abraded or wounded, it may be absorbed and produce serious effects. Some accidents of this kind have occurred in the practice of photography. (*Ann. d'Hyg.*, 1863, vol. 1, p. 454.) A mixture of

cyanide of potassium, cyanide of silver, and chalk, is used under the name of *argentine*, for silvering metallic plates, and as a sort of plate-powder. Dr. Martius met with a case in which a lady suffered some severe symptoms of poisoning owing to her having used this powder for silvering metal by rubbing it on the surface with linen. The powder had penetrated, and had been absorbed through the skin. (Mehu, *Ann. Pharm.*, 1874, p. 220.)

Dr. Chanet has directed attention to the local action of this poison on the hands of the workmen engaged in the arts of electro-gilding and silvering. The hands of these men are almost always covered with ulcers. The skin about the joints is fissured, and an oozing of blood often takes place. The nail with its root participates in the inflammation. The workmen informed him that on dipping their arms in the bath for a few seconds, the whole of the skin became reddened. The ulceration of the soft parts continued even to the bone, producing great pain and broken rest. (*Gaz. des Hôp.*, 24 Juil., 1847, p. 374.) The strong alkalinity of the solution would explain some of these effects, for the solution readily dissolves the cuticle, and exposes the true skin. The excoriations produced may, however, lead to the absorption of the poison, and to all the effects of chronic poisoning by prussic acid.

A patient was directed to use as an injection a solution of rather less than *five grains* (4.6 gr.) of the cyanide dissolved in six ounces and a half of water. He was seized soon afterwards with convulsions, palpitation, slow and difficult respiration, coldness of surface, dilatation of pupils, and fixedness of the eyes. He died in an hour. (*Ann. d'Hyg.*, 1843, vol. 1, p. 412.) It appears that, thirty-six hours previously, he had used a similar injection without injury, but the probability is that the cyanide then employed was not pure.

Fatal Dose and Period of Death.—Two grains and a half (2.44 gr.) of the pure salt are equivalent to one grain of anhydrous prussic, or fifty minims of the London pharmacopœial acid. Hence the cyanide may be regarded as a solid compound of prussic acid containing of this poison, in its most concentrated form, no less than 39.3 per cent. by weight! A dose of from three to five grains of the pure salt may, therefore, easily prove fatal. From a case above related, it would appear that a dose of less than *five grains* has actually destroyed life, and in another case *five grains* proved fatal. Death has taken place in a quarter of an hour, but it may prove even more rapidly fatal.

The cyanide is not used medicinally in England. The *medicinal dose* is estimated at from one-eighth to one-fourth of a grain, but, as the salt is of uncertain composition, it is a most dangerous substance to employ. From its great solvent powers on the metals it is extensively used in the arts of electro-gilding and plating, as well as in photography. The solution is improperly kept exposed, and is constantly evolving prussic acid in vapor.

Treatment.—The symptoms occur with such rapidity and violence, that there is scarcely time to employ treatment. The administration of a weak solution of green sulphate of iron would have the effect of decomposing the poison, and converting it to Prussian blue. Cold

affusion and other remedies used in poisoning by prussic acid should be also applied.

In one instance the iron treatment appears to have been effectual. A photographer swallowed, by mistake, three grains of the cyanide in solution. Having discovered his error, he immediately swallowed a quantity of developing solution (sulphate of iron). Vomiting was excited by mustard. He threw off a quantity of Prussion blue, and recovered. (*Guy's Hosp. Rep.*, 1868, p. 239.)

Analysis.—Cyanide of potassium is usually seen in hard white uncrystalline masses. It has an acrid alkaline bitter taste, and is without any odor until air and moisture have had free access to it. It then emits the well-marked odor of prussic acid. It is deliquescent, and very soluble in water: the solution, when pure, is colorless, and has a strong alkaline reaction, a soapy feel, and a powerful odor of prussic acid. It is not very soluble in pure and strong alcohol. 1. It is decomposed by all acids, and prussic acid is set free. 2. The potash is precipitated by tartaric acid and chloride of platinum. 3. It gives a white precipitate with nitrate of silver, which, when dried and heated, possesses all the properties of cyanide of silver. This precipitate is easily redissolved by a slight excess of the solution of cyanide of potassium. 4. If a solution of green sulphate of iron is added to a solution of the cyanide of potassium, and after agitation, diluted sulphuric acid, Prussian blue will result. 5. A single grain of this salt, moistened with water in a watch-glass, gives a well-marked reaction by its vapor, with the silver and sulphur tests. Should this experiment fail, a drop of the bisulphide of ammonium may be heated with the cyanide, the liquid acidulated with hydrochloric acid, and a solution of persulphate of iron added. The red color of the sulphocyanate of iron is immediately brought out.

Organic Liquids.—The salt may be obtained as a soluble fixed residue from organic matter by drying and incinerating it in close vessels. Any liquid containing it will have the odor of prussic acid. A small portion of the liquid should be filtered or dialyzed for preliminary testing. If the cyanide is present the addition of a solution of green sulphate of iron and hydrochloric acid will produce Prussian blue before distillation. By distilling the organic liquid with diluted sulphuric acid, prussic acid will be obtained in the receiver, and sulphate of potash may be procured by incinerating the residue left in the retort. M. Bonjean found in one experiment that cyanide of potassium was entirely lost as a result of decomposition in the dead body of an animal. After forty days he was unable to detect it, either by the odor or by tests, in the stomach of a rabbit, which had been killed by $\frac{8}{10}$ths of a grain, and into which a like quantity had been introduced soon after death. (*Op. cit.*, p. 33; see also p. 711.) The cyanide of potassium is liable, during putrefaction, to be converted into sulphocyanide. A case of poisoning occurred to Dr. Letheby in September, 1864, in which sulphocyanide of potassium and carbonate of potash were found in the stomach in place of the poison actually taken. In these cases it should then be sought for by digesting the viscera in a mixture of alcohol and water. (See p. 582, *ante.*) The cyanide may be found as an impurity

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Dr. Chanet has directed attention to the local action of this poison on the hands of the workmen engaged in the arts of electro-gilding and silvering. The hands of these men are almost always covered with ulcers. The skin about the joints is fissured, and an oozing of blood often takes place. The nail with its root participates in the inflammation. The workmen informed him that on dipping their arms in the bath for a few seconds, the whole of the skin became reddened. The ulceration of the soft parts continued even to the bone, producing great pain and broken rest. (*Gaz. des Hôp.*, 24 Juil., 1847, p. 374.) The strong alkalinity of the solution would explain some of these effects, for the solution readily dissolves the cuticle, and exposes the true skin. The excoriations produced may, however, lead to the absorption of the poison, and to all the effects of chronic poisoning by prussic acid.

A patient was directed to use as an injection a solution of rather less than *five grains* (4.6 gr.) of the cyanide dissolved in six ounces and a half of water. He was seized soon afterwards with convulsions, palpitation, slow and difficult respiration, coldness of surface, dilatation of pupils, and fixedness of the eyes. He died in an hour. (*Ann. d'Hyg.*, 1843, vol. 1, p. 412.) It appears that, thirty-six hours previously, he had used a similar injection without injury, but the probability is that the cyanide then employed was not pure.

Fatal Dose and Period of Death.—Two grains and a half (2.44 gr.) of the pure salt are equivalent to one grain of anhydrous prussic, or fifty minims of the London pharmacopœial acid. Hence the cyanide may be regarded as a solid compound of prussic acid containing of this poison, in its most concentrated form, no less than 39.3 per cent. by weight! A dose of from three to five grains of the pure salt may, therefore, easily prove fatal. From a case above related, it would appear that a dose of less than *five grains* has actually destroyed life, and in another case *five grains* proved fatal. Death has taken place in a quarter of an hour, but it may prove even more rapidly fatal.

The cyanide is not used medicinally in England. The *medicinal dose* is estimated at from one-eighth to one-fourth of a grain, but, as the salt is of uncertain composition, it is a most dangerous substance to employ. From its great solvent powers on the metals it is extensively used in the arts of electro-gilding and plating, as well as in photography. The solution is improperly kept exposed, and is constantly evolving prussic acid in vapor.

Treatment.—The symptoms occur with such rapidity and violence, that there is scarcely time to employ treatment. The administration of a weak solution of green sulphate of iron would have the effect of decomposing the poison, and converting it to Prussian blue. Cold

affusion and other remedies used in poisoning by prussic acid should be also applied.

In one instance the iron treatment appears to have been effectual. A photographer swallowed, by mistake, three grains of the cyanide in solution. Having discovered his error, he immediately swallowed a quantity of developing solution (sulphate of iron). Vomiting was excited by mustard. He threw off a quantity of Prussion blue, and recovered. (*Guy's Hosp. Rep.*, 1868, p. 239.)

Analysis.—Cyanide of potassium is usually seen in hard white uncrystalline masses. It has an acrid alkaline bitter taste, and is without any odor until air and moisture have had free access to it. It then emits the well-marked odor of prussic acid. It is deliquescent, and very soluble in water: the solution, when pure, is colorless, and has a strong alkaline reaction, a soapy feel, and a powerful odor of prussic acid. It is not very soluble in pure and strong alcohol. 1. It is decomposed by all acids, and prussic acid is set free. 2. The potash is precipitated by tartaric acid and chloride of platinum. 3. It gives a white precipitate with nitrate of silver, which, when dried and heated, possesses all the properties of cyanide of silver. This precipitate is easily redissolved by a slight excess of the solution of cyanide of potassium. 4. If a solution of green sulphate of iron is added to a solution of the cyanide of potassium, and after agitation, diluted sulphuric acid, Prussian blue will result. 5. A single grain of this salt, moistened with water in a watch-glass, gives a well-marked reaction by its vapor, with the silver and sulphur tests. Should this experiment fail, a drop of the bisulphide of ammonium may be heated with the cyanide, the liquid acidulated with hydrochloric acid, and a solution of persulphate of iron added. The red color of the sulphocyanate of iron is immediately brought out.

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SULPHO- AND FERROCYANIDE OF POTASSIUM.

The sulpho- and ferrocyanic acids, whether free or combined with alkalies, are said not to be poisonous; but further experiments are required to determine to what extent they may be noxious to man. A singular case, in which *Sulphocyanic acid* was alleged to have been the cause of death, will be found reported in the *Brit. and For. Med. Rev.*, July, 1839. A man wishing to destroy himself, swallowed a liquid which he had obtained by distilling ferrocyanide of potassium with strong sulphuric acid. He was found dead in his room, and twenty-four hours afterwards the body was examined. The stomach was not inflamed, but part of its mucous surface was softened, and of a brownish-black color. There was no odor of prussic acid. Some doubt having arisen respecting the products of such a distillation, experiments were performed to determine this point; but the results obtained by the different experimentalists did not agree. In repeating the distillation, I have found that prussic acid in large, and sulphocyanic acid in small quantity, were produced; and it is highly probable that death was really caused by prussic acid, which may have been the case, although no odor was perceptible. The blackened state of the stomach was probably due to some strong sulphuric acid being mixed with it.

Sulphocyanic acid and *Sulphocyanide of potassium* have been found, in moderately large doses, to cause the death of animals. Bernard, who has experimented on this subject, states that the sulphocyanide produces direct paralysis of the muscular system, and arrests the action of the heart—that, in fact, it is a cardiac poison; but this was observed only where the poison had been introduced directly into the circulation—a case which is not likely to present itself in medical jurisprudence. A solution of it injected into the stomach or under the skin of a rabbit, produced no symptom of poisoning. (*Leçons sur les Substances Toxiques*, 1857, pp. 351, 355, 386.) Bernard ranks this as a blood-poison of great power. The salt is generally present in small quantity in saliva: hence it must be formed and secreted by the blood. Dr. Leared examined the saliva of fifty individuals, and found sulphocyanide in the greater number. He had detected it in the urine in the proportion of about one-eighth of a grain in sixteen ounces of normal urine, and also in the blood of man and all other vertebrate animals. (*Proc. R. S.*, 1869, No. 114.) This salt, unlike the ferro- and ferricyanides of potassium, is a remarkable deoxidizer, and its noxious operation on the blood may depend on its removing ozone from that fluid.

With respect to the *Ferrocyanide* and the *Ferricyanide* of potassium, Bischoff found that five grains produced tremors in a small rabbit. These passed off; the animal ate its food readily, but died in five days. The ferrocyanide is remarkable for the rapidity with which it is absorbed and eliminated in the urine of animals to which it has been given. (See *ante*, p. 38.) On a charge of poisoning with the ferrocyanide of potassium, which occurred in Germany, the medical witnesses were asked whether it was a poison. They could not answer the question, but said it would undergo a decomposition in the stomach which would render it inert! In this instance there was no proof that the salt had even been swallowed; and the sudden death of the woman appeared to be due to hydrothorax. (Canstatt's *Jahresbericht,* 1844, B. v. s., 291.) Any acids in the stomach would tend to decompose it, and set free prussic acid; but this change has but little tendency to take place at the natural temperature of the body (98°). Dr. Stevenson informed me that he met with a case in which a man took a dose of this salt, and soon afterwards a large dose of tartaric acid. The result was that by a chemical change prussic acid was produced in his stomach, and he died from the effects of this poison. Further experiments are required to determine the properties of this compound. According to Schubarth, it is not poisonous to man or animals in two-drachm doses. (Wibmer, *op. cit.*, vol. 2, p. 356.) It was formerly supposed to contain prussic acid, and that it was therefore highly deleterious. It is now known, however, that prussic acid is not a constituent but a product resulting from a reaction of its elements, and that it has no independent existence in the salt.

Analysis.—Sulphocyanide of potassium is a white crystallizable salt, very soluble in water, and forming with it, a colorless neutral solution. The tests which may be employed are—1. *Persulphate of iron.* If this is added to a solution of the sulphocyanide, even when in small proportion, it immediately produces a deep blood-red color. The red color disappears, and a milky-white precipitate is thrown down on the addition of a solution of corrosive sublimate, or on boiling the liquid either with or without the addition of a mineral acid. It is also destroyed by dilution with water. 2. *Iodic acid.* When added to the solution, iodine is set free, indicated by the blue color produced on the addition of starch, or by shaking the liquid with sulphide of carbon, which dissolves and separates the iodine. The potash may be discovered by the usual tests. When distilled with sulphuric acid it yields a liquid containing prussic acid and sulphuretted hydrogen.

Ferrocyanide of Potassium. — This is a well-known yellow salt, crystallizing in square tables, which are somewhat tough. It is easily dissolved by water, forming a neutral yellow solution. *Persulphate of iron* gives with it, even when considerably diluted, a deep blue precipitate (Prussian blue). When the powder is warmed with diluted sulphuric acid, prussic acid is set free. This may be procured by distillation, or if the salt be in small quantity (one grain), it may be proved to exist by the silver and sulphur tests for prussic acid applied to the vapor.

CYANIDE OF IRON—PRUSSIAN BLUE.

This substance does not appear to possess any poisonous properties. It is said to be much employed, when mixed with a yellow coloring matter, to give a green color to factitious tea leaves. In a seizure which was made of some spurious tea, a question was put by the magistrate—whether Prussian blue was a poison. One of the "experts," who gave evidence, is reported to have said that it was a decided poison: that it consisted of iron, nitrogen, and carbon, and was strongly impregnated with prussic acid! This evidence appears to have been received without any comment. Under the Adulteration Act recently passed, 1873–4, this question has again arisen in a number of cases in which tea-dealers have been charged with selling adulterated tea. It seems that it is the practice of the Chinese to give a green color to tea leaves, and thus to manufacture green tea. China clay or French chalk is also used to give a facing to tea.

The substances here mentioned do not possess any poisonous properties; but undoubtedly the vendor should be bound to inform the purchaser that the tea has been mixed with Prussian blue, clay, etc. With regard to Prussian blue, Schubarth found that two drachms produced no effect whatever on a dog. He therefore contends that it is not a poison, and that it should be regarded as an indifferent substance. (Wibmer, *op. cit.*, vol. 2, p. 356.)

Chemical Analysis.—Prussian blue is a tasteless powder of a deep blue color, insoluble in water, alcohol, and the diluted acids. It may be identified by the following characters: 1. When heated in the air it turns brown and becomes incandescent. 2. If warmed with a few drops of caustic potash, oxide of iron is precipitated, and ferrocyanide of potassium is formed. 3. It is dissolved by strong sulphuric acid at the boiling-point, forming a white liquid (by dehydration), but reacquires a blue color when added to water.

CYANIDES OF MERCURY AND SILVER.

A full account of the poisonous properties of the *cyanide of mercury* has been elsewhere given (*ante*, p. 385). From the observation of its effects on man, it appears to act more like a mercurial poison than a compound of cyanogen. I am not aware that the *cyanide of silver* has ever given rise to any instance of poisoning in the human subject. It it very insoluble in water, but it is nevertheless a noxious substance. Mr. Nunneley found, in his experiments on animals, that it acted on them like hydrocyanic acid, but in a weaker degree. (*Prov. Trans.*, N. S., iii, 86.)

Analysis.—For the analysis of cyanide of mercury see *ante*, p. 385, and for that of cyanide of silver, *ante*, p. 574. It is only necessary to state here that both salts may have their nature determined by the vapor tests for prussic acid (*ante*, p. 575). Thus half a grain (of either salt) put into a watch-glass, and moistened with strong hydrochloric acid, gave the characteristic reactions with the silver and sulphur tests in a few seconds.

CHAPTER LXIII.

POISONING WITH ALCOHOL—ACUTE AND CHRONIC—ALCOHOLISM—SYMPTOMS AND APPEARANCES—ETHER IN LIQUID AND VAPOR—ETHERIZATION—SYMPTOMS AND APPEARANCES—ITS USE AS AN ANÆSTHETIC—HYDRATE OF CHLORAL—SYMPTOMS AND APPEARANCES.

ALCOHOL.

THE only form of poisoning by alcohol which a medical jurist has to encounter is that which arises from the taking of large quantities of spirituous liquors, such as gin, whisky, rum, or brandy. The two last-mentioned compounds contain about fifty-three per cent. by measure of alcohol, while gin and whisky are rather stronger, gin containing as much as fifty-seven per cent.

Symptoms.—In general, the symptoms produced by alcohol come on in the course of a few minutes. There is confusion of thought, with inability to stand or walk, a tottering gait, and giddiness, followed by stupor and coma. Should the person recover from this stage, vomiting supervenes. The insensibility produced by alcohol may not come on until after a certain period, and then suddenly. Sir R. Christison met with an instance in which a person fell into a deep stupor some time after he had swallowed sixteen ounces of whisky; there were none of the usual premonitory symptoms. In another instance, a person may apparently recover from the first effects, and then suddenly become insensible and die convulsed. There is a ghastly or vacant expression in the face, which is sometimes suffused and bloated; the lips are livid, and the pupils are dilated and fixed; if they possess the power of contracting under the influence of light, it is a favorable sign. (See *Lancet*, Jan. 27, 1855, p. 89.) The conjunctivæ are generally much suffused. The breath has an alcoholic odor. The more concentrated the alcohol the more rapidly are the symptoms induced, and they are also more severe in their character. Diluted alcohol commonly produces a stage of excitement before stupor, while in the action of concentrated alcohol, there may be profound coma in a few minutes. The cause of death may be generally traced to congestion of the brain or lungs, or both.

Alcohol may act as a poison by its *vapor*. If the concentrated vapor be respired it will produce the usual effects of intoxication. There is a case on record in which a child two years of age was thrown into an apoplectic stupor by the alcoholic vapor of eau de cologne. In this manner a child might be destroyed, and no trace of the poison found in the stomach. In five years (1863–7) thirty-five deaths are reported to have occurred from alcohol by the acute form of poisoning in England and Wales.

One of the remarkable features of poisoning by alcohol is that a *remission* of the symptoms is by no means unfrequent, and that death sometimes takes place suddenly after some hours or days, when a person appears to have recovered entirely from the effects.

The symptoms arising from apoplexy, from concussion of the brain, or the effects of opium, have been sometimes mistaken for those of poisoning by alcohol, and persons have been wrongly charged with being drunk. With respect to *concussion*, a difficulty can arise only in reference to the more advanced stage of poisoning by alcohol, *i. e.*, in which there is profound coma. Intoxication may in general be easily distinguished by the odor of the breath, for so long as the symptoms continue, the alcohol is eliminated by the lungs. If there should be no perceptible odor of any alcoholic liquid, the presumption is that the symptoms are not due to intoxication. When the alcoholic odor is perceptible, the symptoms may still be combined with the effects of apoplexy or concussion—a fact which can be cleared up only by a history of the case, or a careful examination of the head for marks of violence. In poisoning by opium there will be a strong smell of this drug in the breath, the symptoms come on much more gradually, and are marked by drowsiness and stupor, passing into complete lethargy, with general relaxation of the muscles, and inability to walk. In poisoning by alcohol there is either great excitement some time before the stupor, which comes on suddenly, or the person is found in a state of deep coma a few minutes after having taken the poison. In poisoning by opium the face is pale, and the pupils are contracted; in poisoning by alcohol the face, under excitement, is more commonly flushed, and the pupils are generally dilated. Another fact to be noticed is, that while perfect remissions are rare in poisoning by opium, in poisoning by alcohol a person frequently recovers his senses and dies subsequently. When coma has supervened, the patient may be roused by a loud noise or a violent shock in either case, and it is very difficult under these circumstances to draw a well-marked distinction. The odor of the breath, or an examination of the fluid drawn from the stomach by the pump, may then show which poison has been taken, but the treatment is the same in both cases.

A child, æt. 4, drank a quantity of brandy, it is supposed not less than two or three ounces. He was found lying quite insensible—respiration scarcely perceptible, and the pupils widely dilated. A mustard emetic was given to him, which caused slight vomiting, and temporarily roused him; but he soon fell into a state of insensibility, and remained in this state for three hours. The breathing then became more regular, and the pupils alternately contracted and dilated. In five hours he was better, the effects of the brandy were gradually passing off, and in two days he recovered. (*Lancet*, 1872, vol. 2, p. 66.)

Dr. Stevenson reports the case of a boy who swallowed a large quantity of brandy. When brought to Guy's Hospital he was insensible; the surface was cold, but there was no stertorous breathing. The stomach-pump was employed. The patient remained unconscious for twelve hours. From that time he recovered. (*Guy's Hosp. Rep.*, 1869, p. 268.)

Chronic Poisoning—Alcoholism.—When alcohol has been taken for a long period in the shape of intoxicating drinks, the person suffers from a series of diseases, the characters of which are well marked. The usual effects are irritation of the stomach and intestines, nausea, vomiting, purging, jaundice, cerebral congestion, scirrhus of the stomach, dropsy, diabetes, paralysis, delirium tremens, and insanity. Such persons are subject to sudden death by coma. After death morbid changes are discovered in various organs. The mucous membrane of the stomach presents a deep reddish-brown appearance, with patches of congestion or effusion. The liver is commonly enlarged, and of a lighter color than natural; hence it is called the nutmeg or the drunkard's liver. It is not unusual to find the kidneys in a state of granular degeneration. (See *Med. Times and Gaz.*, July 16, 1853, p. 72.) Of all the common consequences of the abuse of alcoholic liquids, delirium tremens is by far the most frequent. Although generally a result of chronic poisoning, a state analogous to it has been known to supervene rapidly. (*Med. Gaz.*, vol. 38, p. 554.) Delirium tremens is sometimes observed when, after long abuse, alcoholic liquids are suddenly discontinued. In these cases it is the result of the withdrawal of the stimulus, hence the symptoms are often mitigated when the use of alcohol is resumed. Something analogous to this is observed in chronic poisoning by opium and hydrate of chloral.

Appearances.—The stomach has been found intensely congested or inflamed, the mucous membrane presenting in one case a bright red, and in another a dark red-brown color. When death has taken place rapidly, there may be a peculiar odor of spirits in the contents; but this will not be perceived if the quantity taken was small, or many hours have elapsed before the inspection is made. The brain and its membranes are found congested, and in some instances, there is effusion of blood or serum beneath the mucous membrane. In a case observed by the late Dr. Geoghegan, in which a pint of spirits had been taken, and proved fatal in eight hours, black extravasation was found on the mucous membrance of the stomach; but no trace of alcohol could be detected in the contents. (*Dub. Med. Press*, vol. 1, p. 293; also *Ed. Mon. Jour.*, June, 1844.) The action of a strong alcoholic liquid on the mucous membrane of the stomach so closely resembles the effects produced by arsenic and other irritants, as easily to give rise to the suspicion of mineral irritant poisoning. A drawing in the Museum collection of Guy's Hospital furnishes a good illustration of this local action of alcohol. The whole of the mucous membrane of the stomach is highly corrugated, and is of a deep brownish-red color. Of all the liquids affecting the brain, this has the most powerful irritant action on the stomach.

Casper examined, on the fourth day after death, the body of a man who had died from excessive drinking. Cadaveric rigidity was well marked, and there was an absence of putrefaction. The skin was in a state of contraction (cutis anserina). The bloodvessels of the membranes of the brain were congested, and on the right hemisphere there was an extravasation of fluid blood. The great vessels of the chest were filled with dark liquid blood; the lungs were normal; the heart was empty. There was an *odor* of alcohol in the head and chest. In another case, after seven days, there was scarcely any sign of putrefaction. There was congestion of the brain; the blood was dark and fluid, and the cavities of the heart contained only a small quantity. The odor of brandy was perceptible in the head and chest. In a third, on the ninth day after death, the body was comparatively fresh; the inspection was made on the eleventh day. The membranes of the brain were congested; the lungs were œdematous. The right cavities of the heart were strongly distended with dark fluid blood; the left cavities were almost empty. The urinary bladder was distended with urine, apparently from a want of power to pass it. There was no odor of alcohol in any part. (*Ger. Med.*, vol. 1, p. 453.)

A case of alcoholic poisoning of a child, æt. 7, referred to me by Mr. Jackaman, coroner for Ipswich, in July, 1863, will show that the odor may rapidly disappear. A girl was found at four o'clock in the morning lying perfectly insensible on the floor. She had had access to some brandy which she had swallowed from a quartern measure found near her, quite empty. She had spoken to her mother only ten minutes before, so that the symptoms must have come on very rapidly. She was seen by Mr. Adams four hours afterwards. She was then quite insensible, in a state of profound coma, the skin cold and covered with a clammy perspiration. There had been slight vomiting. The child died in twelve hours, without recovering consciousness from the time at which she was first found. On inspection, there was congestion of the brain and its membranes; the heart and lungs were quite healthy. The mucous membrane of the stomach presented patches of intense redness, and in some places is was thickened and softened—portions of it were detached and hanging loosely in the stomach, and there were patches of black extravasation about it, evidently from altered blood. It contained a greenish-colored liquid, but there was *no smell* of brandy in it, neither was this perceptible in the breath of the child, four hours after the alcoholic liquid had been taken. At first it was suspected that arsenic had been administered, but the symptoms were not those of arsenical poisoning, and neither arsenic nor any other metallic irritant was present in the contents of the stomach, but slight traces of the vapor of alcohol were detected by the process described below.

Fatal Dose.—The quantity of alcohol required to destroy life cannot be fixed. It must depend on the age and habits of the person. The smallest quantity known to have proved fatal was in the case of a boy, æt. 7, who swallowed two wineglassfuls of brandy (between three and four ounces). Soon afterwards he was observed to stagger; he was sent to bed, and vomited violently. There was then a remission of the symptoms. He got up and sat by the fire; his head, face, and neck were very red, and he was in a profuse perspiration. Half an hour afterwards, he was found perfectly insensible, strongly convulsed, and the skin cold. He died in about thirty hours. The strength of the alcoholic liquid taken will materially influence a medical opinion in such cases. In a case in which I was consulted in March, 1857, a man drank two bottles of port wine (containing eleven ounces of alcohol) in less than two hours. He speedily became intoxicated and utterly helpless, and died, without rallying, from congestion of the brain and lungs.

In a concentrated form it is probable that from two to six ounces of alcohol would prove fatal.

Absorption and Elimination.—This liquid is rapidly absorbed, diffused, and eliminated; but there is reason to believe that a portion undergoes changes while circulating in the blood. Dr. Percy, many years since, performed experiments on dogs with a view of determining the rate of absorption. Six ounces of alcohol, injected into the stomach, killed a dog in an *hour and ten minutes.* Alcohol was obtained from the brain by distillation. In another experiment of a similar kind, alcohol was detected in the brain, blood, urine, and bile. In a third the animal lived *eight hours* after the injection, and no trace of alcohol could be detected in the brain, blood, bile, or in the contents of the stomach. The whole of it had disappeared.

In the case of a man who had died from drinking a quantity of rum, Drs. Christison and Percy obtained alcohol from the brain by distillation; but in another case, in which a man survived three days, no trace of alcohol was found. According to the observations of Dr. Percy, this liquid is not eliminated to any extent by the urine. He separated it only once from the urine of dogs; and he once obtained evidence of its presence in the distillation of five ounces of human urine. (*Experimental Inquiry on Alcohol in the Brain*, by John Percy, M.D., 1859, p. 61.)

The general conclusion to be drawn from these experiments, is that in acute poisoning by alcohol, this liquid is soon absorbed and eliminated, so that if the person has survived a few days none is likely to be detected in the body. It is remarkable that it should be found combined with or deposited in the substance of the brain; but the experiments of Dr. Percy and Sir R. Christison show that this is the organ in which, after death, the absorbed alcohol is likely to be found.

More recent observations on the absorption and elimination of alcohol have been made by Drs. Parkes, Anstie, and Dupré; but these refer to the use of alcohol for dietetic purposes, and therefore apply to the living body. The experiments of Dr. Dupré prove that alcohol, when taken as wine in non-poisonous doses, is eliminated by the kidneys, lungs, skin, and mucous membrane of the bowels. He has detected it as thus eliminated by distillation, and the application of delicate tests to the distilled liquid; but according to him a minute fraction only of the alcohol taken is eliminated through the kidneys (*Proc. R. S.*, March, 1872, p. 268); and it is admitted that the quantity which passes by the skin and the lungs is also small. What then becomes of the difference? Is it oxidized in the circulation, as some believe? Is it deposited in the brain, as Dr. Percy's experiments would lead us to suppose, or is it lost by evaporation? There are no facts at present to enable us to answer these questions with certainty; and a remark made by Dr. Dupré would throw a doubt upon the accuracy of the statement that alcohol is eliminated by the urine. He states that a substance is found in the urine after six weeks' total abstinence (from alcohol), and even after an abstinence of two years, which gives the reactions ordinarily employed for the detection of small quantities of alcohol. (*Op. cit.*, p. 276.) It will be seen from this that an examination of the urine for alcohol in a living person, might give rise to a fallacious result, and lead to a serious error in diagnosis. Making due allowance for the presence of this substance, and correcting his results by experiments on the air expired from the lungs, and the secretion from the skin, Dr. Dupré believes that ten days may be taken as the period for the entire elimination of alcohol. (*Proc. R. S.*, March, 1872, p. 268.) The examination of the breath exhaled from the lungs gives more reliable results than an analysis of the urine.

Period of Death.—In poisoning by alcohol, death may take place in a few minutes, or not until after the lapse of several days. The shortest fatal case which I have found reported, is that of a man who died after swallowing a bottle of gin for a wager. It occurred in London, in 1839; in a quarter of an hour after taking the gin the man appeared intoxicated; he soon became insensible, and died in *half an hour*, although a large quantity of the spirit had been in the meantime removed by the stomach-pump. In general, if the case proves fatal, death takes place within twenty-four hours. Alcohol, it must be remembered, may destroy life indirectly, *i. e.*, by exciting an attack of congestive apoplexy in those who are predisposed to this disease, and thus a small quantity may accelerate death.

Treatment.—The contents of the stomach should be withdrawn by the pump as speedily as possible. Cold affusion, if the surface be warm, or, as suggested by Sir R. Christison, the injection of cold water into the ears, may serve to rouse a person. Death may take place even when the stomach has been emptied, but this affords commonly the only chance of saving life. The vapor of ammonia may be employed as a stimulant, and bleeding may be resorted to if there should be great cerebral congestion. Bleeding should in any case be employed with great caution, as it is apt to depress the vital powers and diminish the chance of recovery. The electro-magnetic apparatus may be used as in poisoning by opium; but it is necessary to remember that keeping a person roused does not aid recovery, so long as the poison is allowed to remain in the body.

Analysis.—The contents of the stomach in a rapidly fatal case may have the odor of alcohol, or of the alcoholic liquid taken. The odor is not always perceptible, or it may be concealed by other odors. In a case of poisoning with gin, the liquid drawn from the stomach by the pump after seven hours had no odor of that spirit. (See p. 610.)

The whole of the contents of the suspected liquid should be distilled in a water-bath, with a proper condensing apparatus attached. (See Fig. 22, p. 320.) If the liquid has an acid reaction, it should be first neutralized by a solution of carbonate of potash or soda. The watery distillate obtained should be mixed with fused chloride of calcium or anhydrous sulphate of copper in sufficient quantity, and submitted to a second distillation in a smaller retort, by a water-bath. The liquid resulting from this second distillation should be agitated with rather more dry carbonate of potash than it will dissolve, in a small tube provided with a stopper, and allowed to stand. A stratum of alcohol, if present, will, after a time, float on the surface, and may be drawn off by a pipette and examined. *Tests.*—1. Alcohol has a hot pungent taste, a peculiar odor, and is very volatile. 2. Absorbed in asbestos, it burns with a pale blue flame, which deposits no carbon on white porcelain; and when burnt in the mouth of an inverted test-tube containing a few drops of solution of baryta, it produces a well-marked deposit of white carbonate of baryta. Lime-water may be substituted for baryta in this experiment. Carbonic acid and water are the sole products of its combustion. 3. It dissolves camphor. 4. It sets free green oxide of chromium when boiled with a few drops of a saturated solution of bichromate of potash mixed with sulphuric acid. (Dr. Thomson, in *Monthly Jour. Med. Science*, Dec. 1846, p. 412.)

The following method will allow of the detection of a quantity of alcohol too small for separation by the process above mentioned. Make a mixture of strong sulphuric acid and a saturated solution of bichromate of potash; moisten with this mixture a few fibres of asbestos, and inclose them in a glass tube connected with the retort or flask in which distillation is carried on. For this purpose a flask or tube similar to those used for the detection of chloroform vapor will be found serviceable (see Fig. 59, p. 624). The smallest portion of alcohol vapor passing over the asbestos immediately renders it green, by converting the chromic acid to oxide of chromium. This may serve as a trial-test or for evidence, according to circumstances. The tube may be removed, and the condensed vapor collected for the application of the other tests. The vapor of ether and of pyroxylic spirit produce a similar result. This method may be employed in the analysis of the *tissues*, but it cannot be adopted in cases in which sulphuretted hydrogen is present.

From lapse of time, the effects of treatment, or absorption and elimination, there may be no trace of alcohol in the stomach or intestines, nevertheless, the person may have died from the effects. In a case fatal in eight hours, which occurred to the late Dr. Geoghegan, no alcohol was found in the stomach (*ante*, p. 607). One cause of failure may sometimes be traced to the distillation being restricted to only a portion of the contents. It is advisable to distil the *whole*, as, if necessary, the distillate or the residue can be afterwards examined for other poisons.

ETHER.

Symptoms and Effects.—It has been long known that the vapor of ether acts upon the brain and nervous system like a powerful narcotic, but there has been but little experience of its effects as a liquid. In moderate doses, it has a hot burning taste, and produces during swallowing, a sense of heat and constriction in the throat. It causes, like alcohol, great excitement and exhilaration, followed by intoxication, but persons may become habituated to it, and thus after a time it may be taken in very large quantities with comparative impunity. It causes intoxication more rapidly than alcohol, but this state is of shorter duration with ether than it is with alcohol. In the north of Ireland, it is said to have been much used of late years as a substitute for whisky. The effects produced on the system, when a large dose has been taken, are not unlike those produced by alcohol. Orfila found that about half an ounce of sulphuric ether, administered to a dog, caused, in a few minutes, a disposition to vomit. This was followed by giddiness, and in ten minutes by an entire loss of power in the muscles. The breathing was painful and hurried, but there were no convulsions. After a slight abatement in the symptoms, the dog fell into a state of insensibility, and died in three hours. The whole of the mucous membrane of the stomach was of a blackish-red color, and with the other coats intensely inflamed. There was slight inflammation of the duodenum; but the rest of the alimentary canal was in a healthy condition. The heart contained black blood partly coagulated; the lungs were gorged with black blood. (*Toxicol.*, vol. 2, p. 531.) Ether as a liquid has not, so far as I know, destroyed human life; but when its vapor has been breathed for anæsthetic purposes, it has been the cause of death on several occasions. According to some, it is a much safer anæsthetic than chloroform, although the latter is more extensively employed. The medicinal dose of ether is from half a drachm to two drachms. Dr. Buchanan has known seven drachms of it taken at once; it produced at the pit of the stomach an uneasy sensation of heat and pain, which only the callous stomach of a dram-drinker could withstand. (*Med. Gaz.*, vol. 39, p. 715.)

Ether Vapor — Etherization.—When the vapor of this liquid is breathed, it enters the blood in the pulmonary vessels, and its effects are almost immediate. The person falls into a lethargic condition, the breathing becomes slow, deep, and stertorous, the skin pale and cold, the face livid, the lips assume a darker hue, the pulse is quickened, and the muscles of the body are flabby and relaxed. The eye is glassy and the pupils are dilated and fixed, but the late Dr. Snow states that he found the eye sensible to light in all the stages of etherization. A small quantity of ether, introduced into the blood through the lungs, produces these striking symptoms in from *two* to *four minutes;* and if fresh air be substituted as soon as unconsciousness begins, they disappear just as rapidly. In a more advanced stage the pulse slackens, and the temperature of the body rapidly falls. Half an ounce of ether, or even less, inhaled in the form of vapor, would produce a much more powerful effect on the system than one or two ounces taken into the stomach as a liquid. The sudden cessation of the symptoms, and the restoration of sensibility, are owing to the rapid elimination of the volatile vapor through the lungs. If the breathing of the vapor be prolonged for from ten minutes to half an hour, coma ensues, the pulse sinks, and there is some difficulty in rousing the person. The after effects are also more serious: there is exhaustion, a feeling of stupefaction, with other unpleasant narcotic symptoms; but occasionally the patient has fallen into a quiet sleep. The most remarkable effects in those who suffer under this form of poisoning is the apparently complete paralysis of the nerves of sensation; for the most painful operations have been often borne by persons in this state without any consciousness of pain. As a general rule no dangerous effects appear to have followed the breathing of this vapor for surgical purposes; but this inference has been chiefly drawn from those cases in which it had been cautiously administered for a short period; and probably there was no tendency to congestion of the brain or lungs. The narcotic effects are produced in three, and on an average in five minutes. The quantity of liquid ether required to produce complete insensibility by its vapor varies from six drachms to one ounce in the adult, and in children in the same proportion according to their age. The quantity required to keep up insensibility is seldom greater than that which produced etherization.

The vapor of *methylated ether* operates in a similar manner, and it is equally liable to cause death suddenly when administered for surgical purposes. A patient of Mr. Tait's, at the Birmingham Hospital, was about to undergo the operation for ovariotomy. Five drachms of methylated ether in vapor were administered to her on a fold of a towel by the resident medical officer. The pulse suddenly stopped, the pupils became dilated, and respiration ceased. All efforts at restoration were fruitless. On inspection the heart and all the other organs were healthy excepting the ovary. (*Lancet*, July 5, 1873, p. 23.)

According to Dr. Fifield, U. S., who has had a large experience in the administration of ether-vapor for anæsthetic purposes, etherization presents three definitely marked stages: 1, that of muscular relaxation; 2, tetanic convulsive action; 3, complete loss of sensibility, with stertorous or snoring breathing. Unless this stage is reached, there is not full insensibility to pain. (*Brit. Med. Jour.*, March 20, 1875, p. 390.) The great superiority of ether over chloroform as an anæsthetic is shown, in his opinion, by its perfect safety. He states that he has witnessed its operation in more than a thousand cases, and had never seen or heard of a fatal result. This is strong testimony in favor of ether-vapor; but there is no doubt, from cases given below, that it may operate as a poison in the blood and destroy life.

Post-mortem Appearances.—In the case of a man who died in about *ten minutes* from the effects of the vapor, on inspection twenty-two hours after death, the brain, lungs, heart, kidneys, and spleen, when cut into, gave out a strong odor of ether. The blood in the vessels was of a very dark color, liquid, and of a viscid character. The posterior part of the lungs was strongly congested with dark-colored blood; and in the anterior portion of the organs, a frothy mucus was found filling the air-tubes. The mucous membrane of the whole of the air-passages was deeply injected. (*Med. Gaz.*, vol. 41, p. 432.) On examining the bodies of animals thus poisoned, the principal appearances have been great congestion of the vessels of the membranes and of the sinuses of the brain, the substance being but little altered. The vessels of the upper part of the spinal marrow have been observed to be especially distended with dark-colored blood. Both sides of the heart have been found filled with dark blood; the liver and kidneys gorged; the spleen not always congested; the blood black and liquid throughout the body. The cause of death in these cases may be assigned partly to the want of aeration of the blood by oxygen, and its accumulation in this impure state in the brain; and partly to a directly poisonous action of the absorbed vapor, only manifested by its employment for a long period. In order to prevent this, it has been advised to allow the patient to breathe air occasionally, and to alternate the breathing of pure air with that of the vapor; but, unless there is a complete restoration of sensi-

bility and consciousness, the poison must go on accumulating in the system, and if the person be allowed to recover thus completely, it may be regarded as a commencement of its poisonous action, *de novo*. If not thus allowed to recover, he is in danger of sinking under its effects. Experience points to the propriety of withdrawing the vapor altogether in those cases in which the administration of it would require to be protracted for a long period in order to produce narcotic effects. The fact that hundreds have recovered without ill effects during its temporary employment for the extraction of teeth, or similar operations, has of course no bearing on this question. A man may breathe a mixture of carbonic acid or sulphuretted hydrogen with air for a few minutes; but he would die if he was compelled to respire it for half an hour longer. The vapor is so insidious in its operation that it may be respired during natural sleep without rousing the individual, and there is no doubt that it might thus be used as a ready means of destruction for the young and the aged. (*Gaz. Méd.*, Sept. 11, 1847, p. 725.)

In another fatal case the death of a man was occasioned by the breathing of the vapor at intervals for a period of only ten minutes during an operation. He recovered from the comatose effects; but there was no tendency to reaction, and he gradually sank, and died on the second day. It was remarked in this case that there was great flaccidity and general relaxation of the muscular system, and the arteries which were divided during the operation (lithotomy) appeared to have lost all their contractile power. On inspection, there was membranous congestion of the brain; the lungs were engorged at the back part; the heart was flaccid, of its natural size but nearly empty; the left kidney pale, the right congested. The blood was perfectly fluid. (*Med. Gaz.*, vol. 39, p. 414.) A similar case was privately communicated to me by an eminent London surgeon, in which he stated that there could be no doubt of the vapor having been the direct cause of death. The patient sank after the operation, under symptoms which in similar circumstances he had never before witnessed. In the *Medical Gazette*, there is reported another case (vol. 39, p. 585) in which the vapor was administered to a woman, for a period of thirty-five minutes. She recovered her senses, but did not rally from the operation. She complained of numbness in the feet and legs, and the secretions were suspended. She died the following day. On this occasion the vapor appeared to induce a perfect state of paralysis of the brain and nervous system. On inspection, the lungs were slightly congested posteriorly; the heart was flabby, and contained less blood than usual; the brain healthy, its membranes rather congested, the blood generally in a liquid state. In a case which occurred to Mr. Eastment, there was no disease, nor any particular state of the body to account for death. Amputation was performed for compound fracture of the thigh; the ether was inhaled by the patient, a boy æt. 11, for about ten minutes. After the operation he was not only greatly exhausted, but in a state of apparent intoxication. There were alternate manifestations of excitement and depression of the sensorial powers, at one time resembling delirium, at another syncope, and again passing into violent intoxication, until death took place three hours after the operation. (*Med. Gaz.*, vol. 39, p. 632.) The symptoms were here

such as might be expected from the poisonous effects of ether, and unlike those which usually attend collapse from an operation.

These facts, then, show that the respiration of this vapor, even for so short a period as ten minutes, may be in some instances attended with fatal consequences. Whether the vapor was properly administered or not, is, in relation to legal medicine, not so much the question as whether it caused death! In any case the inhalation of this vapor must be looked upon as temporary poisoning, with, *cæteris paribus*, a better chance of recovery than exists in most other instances of aerial poisoning.

Ether is said to produce its narcotic effects, when administered as a vapor by the rectum, without the production of those distressing symptoms which often accompany the first attempts at breathing it. (See *Med. Gaz.*, vol. 39, p. 950.)

Treatment.—In reference to the *vapor*, the failure of the pulse, with stertorous breathing, frothing at the mouth, and great lividity of the face, are signs of danger. The ether should be immediately withdrawn, the face and neck exposed to a free current of air, and cold water dashed upon the skin, although, from the suspension of sensibility, stimulants produce little effect. Artificial respiration should be resorted to when the breathing has ceased.

Analysis.—When ether has been taken as a liquid, it may be separated from the contents of the stomach, if present, by the process described for alcohol (p. 611). The chromic acid process (see Alcohol) applied to the vapor during distillation will enable the analyst to detect a minute quantity, and by its peculiar odor ether may be easily distinguished from alcohol or pyroxylic spirit. 1. Ether is at once identified by its powerful odor, even in the smallest proportion. 2. It is highly inflammable, and burns with a yellow smoky flame, producing carbonic acid and water. When shaken with its bulk of water, only a small portion is dissolved; the rest floats on the surface.

Hoffman's liquor is a mixture of alcohol and ether. This may be easily examined by agitating it with half its bulk of water; the ether (beyond about one-tenth of the quantity of water used) rises to the surface, and may be drawn off by a pipette; the alcohol is dissolved and retained by the water. This liquid may be procured by distillation with carbonate of potash or fused chloride of calcium, and its properties then tested.

Organic Liquids—The Tissues.—When death has taken place from ether-vapor there is a strong odor throughout the body, if the examination is recent. The quantity absorbed by the blood is small, and it is probably partially converted to aldehyde in that liquid. There can be but little hope of success in attempting to procure it from the blood or the soft organs by distillation, although M. Flandin states that he has extracted it from the blood. Whether ether be taken in the form of liquid or breathed as vapor, there is no doubt that it is absorbed and circulated with the blood, in the latter state with great rapidity. M. Amussat noticed, in his experiments on animals, that after long inhalation the arterial blood lost its red color and became black. The bright arterial tint was, however, soon resumed on suspending the process. Ether, besides rendering the blood black, causes it to become

bility and consciousness, the poison must go on accumulating in the system, and if the person be allowed to recover thus completely, it may be regarded as a commencement of its poisonous action, *de novo*. If not thus allowed to recover, he is in danger of sinking under its effects. Experience points to the propriety of withdrawing the vapor altogether in those cases in which the administration of it would require to be protracted for a long period in order to produce narcotic effects. The fact that hundreds have recovered without ill effects during its temporary employment for the extraction of teeth, or similar operations, has of course no bearing on this question. A man may breathe a mixture of carbonic acid or sulphuretted hydrogen with air for a few minutes; but he would die if he was compelled to respire it for half an hour longer. The vapor is so insidious in its operation that it may be respired during natural sleep without rousing the individual, and there is no doubt that it might thus be used as a ready means of destruction for the young and the aged. (*Gaz. Méd.*, Sept. 11, 1847, p. 725.)

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Organic Liquids—The Tissues.—When death has taken place from ether-vapor there is a strong odor throughout the body, if the examination is recent. The quantity absorbed by the blood is small, and it is probably partially converted to aldehyde in that liquid. There can be but little hope of success in attempting to procure it from the blood or the soft organs by distillation, although M. Flandin states that he has extracted it from the bloo[illegible] Whether ether be taken in the form of liquid or brea[illegible] apo[illegible] is no doubt that it is absorbed and [illegible] latter state with great rapidity. M. [illegible] s on animals, that after long inha- [illegible] ed color and became black. The [illegible] on resumed on suspending the pro- [illegible] blood black, causes it to become

bility and consciousness, the poison must go on accumulating in the system, and if the person be allowed to recover thus completely, it may be regarded as a commencement of its poisonous action, *de novo*. If not thus allowed to recover, he is in danger of sinking under its effects. Experience points to the propriety of withdrawing the vapor altogether in those cases in which the administration of it would require to be protracted for a long period in order to produce narcotic effects. The fact that hundreds have recovered without ill effects during its temporary employment for the extraction of teeth, or similar operations, has of course no bearing on this question. A man may breathe a mixture of carbonic acid or sulphuretted hydrogen with air for a few minutes; but he would die if he was compelled to respire it for half an hour longer. The vapor is so insidious in its operation that it may be respired during natural sleep without rousing the individual, and there is no doubt that it might thus be used as a ready means of destruction for the young and the aged. (*Gaz. Méd.*, Sept. 11, 1847, p. 725.)

In another fatal case the death of a man was occasioned by the breathing of the vapor at intervals for a period of only ten minutes during an operation. He recovered from the comatose effects; but there was no tendency to reaction, and he gradually sank, and died on the second day. It was remarked in this case that there was great flaccidity and general relaxation of the muscular system, and the arteries which were divided during the operation (lithotomy) appeared to have lost all their contractile power. On inspection, there was membranous congestion of the brain; the lungs were engorged at the back part; the heart was flaccid, of its natural size but nearly empty; the left kidney pale, the right congested. The blood was perfectly fluid. (*Med. Gaz.*, vol. 39, p. 414.) A similar case was privately communicated to me by an eminent London surgeon, in which he stated that there could be no doubt of the vapor having been the direct cause of death. The patient sank after the operation, under symptoms which in similar circumstances he had never before witnessed. In the *Medical Gazette*, there is reported another case (vol. 39, p. 585) in which the vapor was administered to a woman, for a period of thirty-five minutes. She recovered her senses, but did not rally from the operation. She complained of numbness in the feet and legs, and the secretions were suspended. She died the following day. On this occasion the vapor appeared to induce a perfect state of paralysis of the brain and nervous system. On inspection, the lungs were slightly congested posteriorly; the heart was flabby, and contained less blood than usual; the brain healthy, its membranes rather congested, the blood generally in a liquid state. In a case which occurred to Mr. Eastment, there was no disease, nor any particular state of the body to account for death. Amputation was performed for compound fracture of the thigh; the ether was inhaled by the patient, a boy æt. 11, for about ten minutes. After the operation he was not only greatly exhausted, but in a state of apparent intoxication. There were alternate manifestations of excitement and depression of the sensorial powers, at one time resembling delirium, at another syncope, and again passing into violent intoxication, until death took place three hours after the operation. (*Med. Gaz.*, vol. 39, p. 632.) The symptoms were here

such as might be expected from the poisonous effects of ether, and unlike those which usually attend collapse from an operation.

These facts, then, show that the respiration of this vapor, even for so short a period as ten minutes, may be in some instances attended with fatal consequences. Whether the vapor was properly administered or not, is, in relation to legal medicine, not so much the question as whether it caused death! In any case the inhalation of this vapor must be looked upon as temporary poisoning, with, *cæteris paribus*, a better chance of recovery than exists in most other instances of aerial poisoning.

Ether is said to produce its narcotic effects, when administered as a vapor by the rectum, without the production of those distressing symptoms which often accompany the first attempts at breathing it. (See *Med. Gaz.*, vol. 39, p. 950.)

Treatment.—In reference to the *vapor*, the failure of the pulse, with stertorous breathing, frothing at the mouth, and great lividity of the face, are signs of danger. The ether should be immediately withdrawn, the face and neck exposed to a free current of air, and cold water dashed upon the skin, although, from the suspension of sensibility, stimulants produce little effect. Artificial respiration should be resorted to when the breathing has ceased.

Analysis.—When ether has been taken as a liquid, it may be separated from the contents of the stomach, if present, by the process described for alcohol (p. 611). The chromic acid process (see Alcohol) applied to the vapor during distillation will enable the analyst to detect a minute quantity, and by its peculiar odor ether may be easily distinguished from alcohol or pyroxylic spirit. 1. Ether is at once identified by its powerful odor, even in the smallest proportion. 2. It is highly inflammable, and burns with a yellow smoky flame, producing carbonic acid and water. When shaken with its bulk of water, only a small portion is dissolved; the rest floats on the surface.

Hoffman's liquor is a mixture of alcohol and ether. This may be easily examined by agitating it with half its bulk of water; the ether (beyond about one-tenth of the quantity of water used) rises to the surface, and may be drawn off by a pipette; the alcohol is dissolved and retained by the water. This liquid may be procured by distillation with carbonate of potash or fused chloride of calcium, and its properties then tested.

Organic Liquids—The Tissues.—When death has taken place from ether-vapor there is a strong odor throughout the body, if the examination is recent. The quantity absorbed by the blood is small, and it is probably partially converted to aldehyde in that liquid. There can be but little hope of success in attempting to procure it from the blood or the soft organs by distillation, although M. Flandin states that he has extracted it from the blood. Whether ether be taken in the form of liquid or breathed as vapor, there is no doubt that it is absorbed and circulated with the blood, in the latter state with great rapidity. M. Amussat noticed, in his experiments on animals, that after long inhalation the arterial blood lost its red color and became black. The bright arterial tint was, however, soon resumed on suspending the process. Ether, besides rendering the blood black, causes it to become more liquid. The change in this fluid is very much like that which is observed in fatal cases of asphyxia. (*Gaz. Méd.*, Sept. 11, 1847, p. 725.)

HYDRATE OF CHLORAL.

This is a solid crystalline substance, produced by the reaction of chlorine on absolute alcohol, and the subsequent addition of a small quantity of water. It has been much used of late as a substitute for opium, and in doses of twenty to thirty grains it has been found to operate as a sedative and narcotic without producing excitement. It has been given in very large doses, sometimes with benefit, but at other times causing dangerous symptoms, followed by sudden death. Medical men who have taken it incautiously have died from its effects. Two instances of this kind are reported in the *Med. Times and Gaz.* (1871, vol. 1, p. 367). The deaths have been frequently sudden, and no remarkable symptoms have preceded dissolution. The person has passed at once from sleep into death.

Symptoms and Appearances.—Hydrate of chloral, when given in proper medicinal doses, produces, after a short interval, a deep sleep, and, when carried far enough, a loss of consciousness, of muscular power, and sensibility. In the chloral-sleep, the pupils are generally contracted, but they dilate on the person awaking. In opiate poisoning they remain permanently contracted. The symptoms of poisoning have varied. There is diminished frequency of respiration, redness of the conjunctivæ, with red and swollen eyelids; a contracted state of the pupils, and a falling of the lower jaw. The pulse has varied much in its character. In cases likely to prove fatal the sleep is heavy; the person cannot be roused; the face is livid or bloated, sometimes flushed; the lips are blue and swollen, the fingers livid, the respiration is deep and sighing or stertorous, the pulse barely perceptible; the pupils in this stage are dilated and insensible to light. Dr. C. Browne noticed among the symptoms, after several medicinal doses had been taken, a flushed condition of the head and face, slight contraction of the pupils, injection of the conjunctivæ, and excitement of the circulation, continuing for about an hour. The patients have complained of burning heat in the face, of a sense of giddiness, inability to walk straight, headache, double vision, and confusion of thought. In one case, the redness of the skin passed into a condition resembling urticaria. In other instances purpura appeared to be a result. One case proved suddenly fatal by causing paralysis of the heart. (*Lancet*, 1871, vol. 1, p. 440.) The late Dr. Fuller, of St. George's Hospital, met with a case in which thirty grains on two occasions produced violent excitement, with delirium, and a sense of oppression and burning at the chest, followed by collapse and an almost entire failure of the heart's action. He had prescribed chloral in doses of from ten to forty-five grains in many cases without any ill effects following. Instances of the fatal operation of this substance are now very numerous. (*Med. Times and Gaz.*, 1870, vol. 2, p. 435, and 1871, vol. 1, p. 132; *Lancet*, 1870, vol. 2, p. 402; 1871, vol. 2, p. 466.) In the fatal cases which have occurred the principal *appearances* noted were a congested state of the vessels of the brain and its membranes.

Chronic Poisoning.—As *chronic* effects produced by chloral when taken for a long period in medicinal doses, Dr. Smith points out an erythematous inflammation of the skin of the fingers, with desquamation and ulceration around the borders of the nails. (*Lancet*, 1871, vol. 2, p. 466.) The cerebral functions are impaired; with loss of intelligence and memory there has been partial paralysis of the limbs. (*Lancet*, 1873, vol. 1, pp. 640, 695.) This effect on the brain may give rise to an important question, viz., whether the long-continued use of chloral may not so affect the brain as to produce a diseased condition, and render a man irresponsible for his acts. Does the sudden withdrawal of it after long use, as with alcohol, produce a condition resembling delirium tremens. Facts are wanting to settle this question decidedly in the affirmative. A case occurred in Canada, in 1873, in which a gentleman was charged with the murder of his wife. He had been accustomed to take chloral in large doses for some weeks. This was withdrawn suddenly. He then became irritable and unnaturally violent, and in a fit of passion, for some trivial cause, threw a petroleum lamp at his wife. This led to the ignition of her dress, and death by burning. He had been up to that time an affectionate husband, and he appeared to have been hardly conscious of the act that he committed. Dr. Tuke and I were consulted on this case, and we gave our opinion that the long-continued use of chloral might have produced a diseased condition of brain, which by the sudden withdrawal of the narcotic might have rendered the accused *pro tempore* irresponsible for his actions.

Fatal Dose.—The average medicinal dose of chloral by the mouth is from twenty to thirty grains, and by hypodermic injection fifteen grains. In the latter case it has sometimes produced pain and swelling of the injected parts. A medicinal dose has in some cases operated as a poison. A woman, æt. 20, died in thirty-five hours from a dose of thirty grains; but persons have recovered from doses of 150 and 160 grains. (*Med. Times and Gaz.*, 1871, vol. 1, p. 169.) A slight overdose may cause sudden death by syncope (*Lancet*, 1873, vol. 1, p. 640), and ordinary doses, long continued, may seriously affect mind and body (*Lancet*, 1873, vol. 1, p. 789). The suddenness of death is quite remarkable in some of these cases. Dr. N. B. Smith, U. S., states that a man who had been in the habit of taking it, and who on the day of his death had purchased three drachms, was found dead in his chamber twenty minutes after he had entered it. The condition of the body and bed-clothes showed that he had died while in the act of stepping into bed. In November, 1872, a man took two doses of 100 grains each. He was found dead in bed, holding an empty vial in his hand. The mucous coat of the stomach was highly congested; but, as the man was an habitual drunkard, this could hardly be ascribed to the effects of chloral. In these cases there has probably been death by syncope from a sudden failure of the heart's action.

Treatment.—As hydrate of chloral is generally taken in the liquid form, the stomach-pump should be used, and the stomach well washed out with tea or coffee. The further treatment for rousing the patient may be similar to that adopted in poisoning by opium or chloroform vapor. Small doses of strychnia have been advised for the treatment of the after-effects.

Analysis.—The hydrate of chloral is a white, brittle, crystalline solid, of a peculiar odor, and a pungent bitter taste. When heated on platinum it melts, and is entirely volatilized without combustion, unless turned into the flame. The hydrate, unlike the alcoholate, is not inflammable. Heated in a close tube, it melts, and does not rapidly solidify. It is distilled over in a liquid form, and after a time it sets into groups of crystals in the glass tube. The hydrate is soluble in water, which retains it on cooling, while the alcoholate is again in great part deposited. The solution is not acid, has no bleaching properties, and gives only a faint milkiness on boiling with a solution of nitrate of silver. When boiled with chloride of gold or nitrate of silver, and potash is added, gold or silver is immediately precipitated. It decomposes a salt of copper like grape-sugar, but in the cold, potash does not redissolve the precipitated oxide of copper. It is dissolved by strong sulphuric and nitric acids, without any change of color. Potash added to the solution while boiling, converts it instantly into chloroform, which escapes with copious effervescence, and may be recognized by its odor, and into formic acid, which combines with the alkali. According to Attfield, 100 parts will yield 82 parts of chloroform. On boiling it with potash, the solution, if the hydrate is pure, acquires only a slight yellow color.

Organic Liquids.—It is by this conversion that hydrate of chloral may be detected in the contents of the stomach. The liquid should be rendered alkaline with potash, and the mixture heated in a flask by a water-bath. The vapor which escapes may be tested for chloroform by the process described at p. 624. Dr. Procter, of York, informs me that he thus detected it in a case of suicidal poisoning. It has been detected as chloroform in the blood of dogs to which it had been administered. (Wiggers, *Jahresbericht*, 1851, p. 566.)

CHAPTER LXIV.

CHLOROFORM—ITS ACTION AS A POISON IN THE LIQUID STATE—SYMPTOMS AND APPEARANCES—CHLOROFORM VAPOR—USE AS AN ANÆSTHETIC—COMPARED WITH ETHER—POISONOUS EFFECTS—APPEARANCES—TREATMENT—ANALYSIS—BICHLORIDE OF METHYLEN—AMYLEN.

CHLOROFORM.

Symptoms and Appearances.—This liquid, when taken in a large dose, appears to produce a species of intoxication, and to affect the system like alcohol. As a liquid it cannot be regarded as an active poison. A case was communicated to me by Mr. Jackson, of Sheffield, in which a man swallowed *four ounces* of chloroform. He was able to walk for a considerable distance after taking this dose, but he subsequently fell into a state of coma; the pupils were dilated, the breathing was stertorous, the skin cold, the pulse imperceptible, and there were general convulsions. He recovered in five days. (*Med. Gaz.*, vol. 47, p. 675.) A private in a cavalry regiment in the United States swallowed nearly two ounces of chloroform. He was seen ten or fifteen minutes afterwards; he had already vomited, and was found insensible, with stertorous breathing, and a pulse of about 60. The stomach-pump was employed, and some spirits of ammonia were injected. The pulse became more feeble, the breathing slower, and the pupils were insensible to light. The surface was cold, and for a time he continued to get worse, the face becoming purple, while the pulse was intermittent and hardly discernible. Two hours and a half after taking the poison, however, a gradual improvement commenced, but sensibility did not return until four hours later. For several days he continued to suffer from great irritability of the stomach, and eventually he had an attack of jaundice. (*Med. Times and Gazette*, November 28, 1857, p. 558.) A man, æt. 42, swallowed two ounces of liquid chloroform, and he died in about six hours afterwards. In this case the pupils were fully dilated, the breathing was stertorous, and the skin covered with a cold perspiration. He rallied for a short time and then sank again, his lips becoming of a dark purple color and his face livid. On inspection the lungs were found much engorged with blood, and there were some apoplectic effusions in these organs. The stomach was slightly inflamed in patches, and the mucous membrane was softened. It contained a dark fluid smelling strongly of chloroform. (*British Med. Jour.*, May, 1866, and *Amer. Jour. Med. Sci.*, October, 1866, p. 571.)

In two cases alarming symptoms were produced by much smaller doses, and one of these proved fatal. A lady swallowed half an ounce of pure chloroform. In five minutes she was quite insensible, generally convulsed, the jaws clenched, the face slightly flushed, the pulse full and rather oppressed, and she foamed at the mouth. She vomited, and in twenty minutes the convulsions had left her; soon afterwards she had a relapse, and did not recover for twenty-four hours. (*Med. Times and Gaz.*, December 12, 1857, p. 615.) The symptoms in this case appear to have been intermixed with those of hysteria and epilepsy. In another case a lady also took half an ounce of liquid chloroform. An emetic was given, and in a few minutes a large quantity of liquid was thrown off the stomach. In about an hour the patient became suddenly livid and then blanched in the face. The pulse was thready and scarcely perceptible; the breathing slow, and after a time stertorous; the hands and face were purple, the eyes were deeply suffused and the pupils were dilated. There was mucous vomiting at intervals. The patient was quite insensible; the eyes were fixed, and the face flushed. She appeared to be dying, but under treatment these symptoms passed away, and in about two hours sensibility returned, and she recovered. (*Lancet*, 1870, vol. 1, p. 290.) A physician, æt. 57, swallowed *three ounces* of chloroform. He immediately began to stagger as if intoxicated. He vomited and sank into a deep stupor, and was in a state of complete anæsthesia. His skin was pale and tolerably warm; the muscles were relaxed, the breathing short, and the action of the heart weak and intermittent. In about fourteen hours sensibility returned. Acute gastritis ensued with rapid collapse, and this proved fatal twenty-nine hours from the time the chloroform was taken. (*Amer. Jour. Med. Sci.*, January, 1870, p. 276.) The following case was communicated to me by Mr. Thursefield, of Broseley, in March, 1854. A boy, æt. 4, was brought to him by his father in a state of total insensibility. It appears that he had swallowed a *drachm* of chloroform, and soon afterwards laid his head in his mother's lap and lost all consciousness. Mr. Thursefield saw him about twenty minutes afterwards. He was then insensible, cold, and pulseless. Mustard plasters were applied to the legs; they acted well, but produced no impression on the sensibility. His breathing varied; it was sometimes natural, at other times stertorous. He became warmer, his pulse full and regular; and he continued *three hours* in this state, when he died quite calmly, without a struggle, in spite of every effort made for his recovery. This is the smallest dose of liquid chloroform that has destroyed human life. The medicinal dose for an adult is from three to ten minims.

Chloroform Vapor—Chloroformization.—The vapor, if respired in a concentrated form, proves speedily fatal to life. When diluted with a certain proportion of air it produces insensibility (*anæsthesia*), with entire loss of muscular power, and the patient rapidly recovers after the vapor is withdrawn. According to the report of a committee, the average proportion of vapor, to act safely as an anæsthetic, is three and a half per cent., the maximum being four and a half per cent. The proportion should be only slowly and never suddenly increased, and it should never be given after a long fast or in the sitting or erect posture. (*Ed. Monthly Jour.*, 1864.) The source of danger on these occasions is sudden death from failure of the heart's action (syncope).

From two to ten minutes are required to produce insensibility; but the time for its production varies with age, temperament, and habit. Dr. Stevenson found that in more than 200 cases of its administration at Guy's Hospital, adults were not usually rendered insensible until after the lapse of eight minutes, the average dose of the liquid given in vapor being three and a half drachms, administered in half drachms. On some persons, as a result of idiosyncrasy, it seems to produce scarcely any effect. In March, 1863, a woman, æt. 40, took ten drachms in thirty-three minutes, in half-drachm doses, and was still conscious and able to talk. These facts show that there is no truth in the statement sometimes made, in cases of alleged robbery or rape, that the person assaulted was rendered suddenly insensible and unable to offer resistance. Chloroform vapor does not produce immediate insensibility unless it also produces complete asphyxia and death. There is, however, one case in which it might be used to aid the perpetration of crime. If the person is already asleep, the application of the vapor might intensify this, and render him or her powerless; but the conditions for thus using chloroform criminally can rarely present themselves. (*Ann. d'Hyg.*, 1874, vol. 1, p. 183.) The late Dr. C. Kidd, who had had much experience in the administration of chloroform vapor, has especially called the attention of the profession to this subject. (*Chloroform in its Medico-legal Bearings*, 1870.) There can be no doubt that false charges of rape have been made against medical men and dentists under the alleged use of this vapor. In general a cross-examination of the prosecutrix alone has been sufficient to show the falsehood of the charge.

Symptoms.—There are considered to be four stages in the administration of the vapor. In the first the patient becomes excited; in the second he talks incoherently, and sensibility is diminished; in the third he is unconscious, but the muscles are rigid; in the fourth the muscles are completely relaxed, and the patient is perfectly insensible. Danger commences with the third stage.

The symptoms produced by the vapor are similar to those produced by the vapor of ether; but the person passes more rapidly into a full state of insensibility, with stertorous breathing. From being at first excited, he becomes motionless; the pupils are at first contracted and then become widely dilated; the sphincters lose their contractile power; the face is pale, sometimes livid; the lips are congested, the breathing is slow, after a time stertorous, the skin cold, and the pulse gradually sinks. There is an entire loss of sensibility. The sinking of the pulse in some cases is so rapid as to expose the patient to sudden death by syncope. In some instances violent convulsions have supervened, even when the dose has been only from half a drachm to a drachm. These effects may be occasionally aggravated by idiosyncrasy, or by latent disease of the heart or brain. The fatal effects of the vapor are likely to be manifested when it is breathed rapidly and unmixed with air. There is no doubt that in a concentrated state, it is a powerful cerebral poison. It is absorbed into the blood, which it darkens, as in asphyxia, and is circulated throughout the system. The blood is probably directly poisoned by it. The effects produced by this vapor come on insidiously; a person who breathes it in the absence of assistance, is thus rendered powerless, and may die with the mouth over the inhaler. Several deaths of this accidental kind are recorded.

Appearances.—Congestion of the vessels of the brain and its membranes has been met with, but not uniformly; the lungs are congested or in an apoplectic condition; the heart flaccid, and the cavities empty or containing but little blood. The right cavities contain more than the left, and they have sometimes been found much distended. (*Ed. Monthly Jour.*, 1864, p. 186; Husemann's *Jahresbericht*, 1872, p. 502.) The blood is in general dark in color, very fluid, and air-bubbles have been in some cases found in it. The muscles of the body are dark-colored. The late Dr. Snow published the results of thirty-four inspections. In three, the state of the lungs is not mentioned; in four, they are said to have been normal, and in twenty-seven, there was engorgement of the lungs, or of the right side of the heart. In the majority of cases both these conditions were met with. (*Med. Times and Gazette*, Oct. 23, 1858, p. 431.) Congestion of the liver, spleen, and the kidneys is not unusual. (See *Ed. Med. Jour.*, Dec. 1855, p. 524.)

Fatal cases have been proportionally much more numerous from the use of chloroform vapor than from ether vapor. In some of these, latent morbid conditions of the heart or brain may have led to the unfortunate result—in others, the improper mode of administering the vapor. But there have been undoubtedly cases in which, with the exercise of proper skill and care, death has still occurred in persons having no disease of the heart or brain.

Thus its fatal operation has been in some cases manifested after the withdrawal of the vapor, apparently from an accumulative effect upon the blood. The patient has become faint and sick, and the action of the heart has suddenly stopped. In one case, witnessed by a friend, the heart suddenly ceased to beat four minutes after the vapor had been withdrawn. The digital arteries, which had been divided in the surgical operation, ceased to bleed. The man was dead. Two fatal cases are reported in the *Brit. Med. Jour.*, for August, 1873, p. 230. In one, a man in good general health died suddenly after having inhaled *one drachm* in vapor. Fatal syncope came on after the chloroform had been withdrawn. In the other case, a lady died at Brighton, under the effects of chloroform, while having a tooth extracted. In this case, it is said, there was fatty degeneration of the heart. It is to this condition of a fatty or flabby heart that the fatal effects are usually ascribed. Assuming this to be to some extent the true cause of the fatality, it must be admitted that fatty and flabby hearts have become exceedingly common since the introduction of chloroform-vapor for surgical and other purposes! But this theory is not necessary to explain the fatal results. They are simply cases of poisoning. In January, 1866, a healthy man died in three minutes from the effects of two drachms of chloroform given in vapor. This death occurred on the operating-table of a London hospital—the vapor having been administered by a gentleman who had given it previously to 300 or 400 patients. Death was sudden, and took place after some deep inspirations and expirations had been made. It was on this occasion candidly admitted that the body was quite healthy. In this and similar cases, either the chloroform had been taken in too concentrated a form too rapidly, or there may have been an idiosyncrasy in the patient to its narcotic poisonous action.

I have been unable to procure any reliable information respecting the statistics of death from chloroform in surgical operations. Hospital authorities are unwilling to place their fatal cases before the public, and all that I can say from inquiry is that the deaths are numerous, although, compared with the total number of cases in which chloroform vapor is used, they are small.

It is a matter for deep regret that neither the greatest care in administration, in regulating the quantity administered, or in the preliminary examination of the person as to his state of health, has sufficed to prevent the occurrence of these fatal cases. In some of them the operation, too, has been of the most trivial kind, and, except at the demand of the patient, the use of such a dangerous agent was not justifiable. As Mr. Brown has pointed out, many of these deaths are unrecorded, for an inquest is not held in every case. (*Brit. Med. Jour.*, July 19, 1873, p. 58.) In the same journal other fatal cases are noticed. (Aug. 23, 1873, p. 230, and Dec. 1872. See also *Lancet*, 1871, vol. 1,

p. 117, vol. 2, pp. 108, 764.) The editors state that in five months there had been in hospital practice ten deaths from chloroform and two from methylen.

The fatal cases have become so numerous that Dr. M. Sims has recently advised the giving up of the use of chloroform-vapor, except in obstetric practice. (*Brit. Med. Jour.*, August, 1874, p. 241.)

As in alcoholic poisoning, a person may die from *secondary* causes after an apparent recovery from the immediate effects of the vapor. (Kesteven's Quarterly Report on Toxicology, *Med.-Chir. Rev.*, April, 1854, p. 582. Case by Dr. King, in *Ed. Med. and Surg. Journal*, January, 1854.) Another case of death subsequent to apparent recovery has been reported by Mr. Lane, surgeon to the Lock Hospital. (*Med. Times and Gaz.*, June 3, 1854, p. 572; *L'Union Medicale*, September 3, 1857, No. 106.)

In the case of a gentleman, æt. 50, one drachm and a half of chloroform was given in vapor for the purpose of reducing a dislocation. The patient struggled while inhaling it. In about four minutes he was under its influence, when dangerous symptoms suddenly appeared. The teeth became firmly clenched, so that artificial respiration was performed with difficulty. After forty minutes' trial the heart's action was good, and respiration fairly restored, but the breathing and the pulse suddenly ceased. (*Lancet*, 1871, vol. 1, p. 703.)

In *Lambe* v. *Barton*, tried at the Wicklow Summer Assizes, 1873, a widow sought to make a medical man responsible for the death of her husband under chloroform. Deceased had met with an accident to his toes, and amputation was considered necessary. He made no objection to take chloroform, and it was administered with proper caution. The man became excited, but in a few minutes the heart suddenly failed, and the man died before the operation could be performed. The rapid death was attributed to "some peculiarity in the patient's constitution which caused the chloroform to produce paralysis of the heart." This could not have been discovered either before or after the administration. The jury found for the defendant. (*Brit. Med. Jour.*, August, 1873, p. 123.)

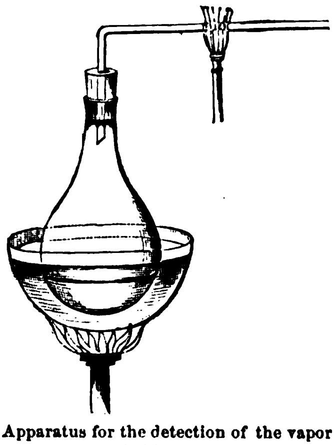
The medical evidence for the plaintiff was to the effect that no examination was made beforehand to ascertain whether the man had or had not disease of the heart. The witnesses had not, however, the candor to state to the Court that such an examination could not have altered the facts, since there are several cases now on record which prove that persons may die in a few minutes from the effects of chloroform vapor, irrespective of the existence of heart disease. This has too frequently been advanced as a theoretical explanation of those fatal cases which were most probably due to direct poisoning by the vapor.

In one instance the vapor proved fatal indirectly by causing suffocation. The case occurred at the London Hospital in 1871. The vapor had been administered to a youth, æt. 13, for an operation to remove squinting. In recovering from the effects of the chloroform, the deceased made an effort to vomit, and at the same time made a deep inspiration. The contents of the stomach were thus drawn into the bronchial tubes, and caused death by suffocation. (*Lancet*, 1871, vol. 2, p. 490.)

From a comparison of results it has been stated that ether vapor is eight times as safe as chloroform; in other words, that seven out of every eight deaths might be avoided if pure ether were used instead of chloroform. (*Brit. Med. Jour.*, December, 1872.) Chloroform vapor destroys life without warning, but ether vapor gives warning when life is threatened, and therefore affords a better chance of recovery. The vapor of a mixture of the two liquids has been employed, and such a mixture is often sold under the name of bichloride of methylen, but this compound vapor has also destroyed life. (*Brit. Med. Jour.*, December, 1873, p. 692; *Med. Times and Gaz.*, August 22, 1857, p. 198.)

Analysis.—Chloroform is a heavy colorless liquid (sp. gr. 1.484), neutral in its reaction, sinking in water in globules, and only to a slight extent dissolving in that liquid. It has a fragrant odor, like that of apples. It is dissolved by alcohol and ether. A solution in alcohol in the proportion of one part by measure to nineteen parts of rectified spirits forms the *Spiritus Chloroformi*, or as it is commonly called, *Chloric Ether*. There is also a tincture, in which the proportion of chloroform is one volume in ten volumes of spirit. Chloroform, although a very dense liquid, is highly volatile, but, unlike ether, its vapor is not inflammable and not readily combustible. Like alcohol, it dissolves camphor readily. Nitric and sulphuric acids produce no change in it. It boils at 140°, and evolves a vapor which at a red heat is resolved into chlorine and hydrochloric acid. On this effect a process has been suggested for separating it from the *blood and tissues*, when it has proved fatal in the form of vapor.

FIG. 59.



Apparatus for the detection of the vapor of chloroform.

The organic liquid supposed to contain chloroform is placed in a flask, like that shown in the annexed illustration (Fig. 59). The neck of the flask is fitted with a cork perforated to admit a hard glass tube, bent at right angles, and having a length of from twelve to fifteen inches. The flask, with its contents rendered slightly alkaline, is gradually plunged into the water at about 160°, and at the same time the middle portion of the tube is heated to full redness by an air-gas jet. At a red heat chloroform vapor is decomposed, and chlorine and hydrochloric acid are among the products of its decomposition. Litmus-paper applied to the mouth of the tube is reddened; starch-paper wetted with iodide of potassium is rendered blue, and nitrate of silver on glass is precipitated white. Two drops of pure chloroform were thus readily detected, and so persistent was the vapor in the closed vessel, that it was detected after one, two, and even three weeks. Two drops added to a quantity of putrefied blood were detected by a similar process after a fortnight, the flask being closed, but the mouth of the tube remaining exposed to air. This method of detecting chloroform by its products appears to be quite satisfactory. In practice, however, it will be found a very difficult matter to detect it, even where we know it has been administered. Some years since, in conjunction with the late Dr. Snow, I examined by this process the blood of a boy who had died in Guy's Hospital from the effects of chloroform vapor, but without detecting any trace of it. There was no odor in the blood, and the result was negative. In 1863 I examined the blood of three persons, taken while they were under the full operation of chloroform, and collected in closely-stopped glass bottles. One of the samples, examined within half an hour after removal from the living body, had no odor of chloroform, and gave not the slightest indication of its presence by the above process. The quantity present in a few ounces of blood may have been too small for detection, or it may have been lost by its volatility. The two other samples, kept in close bottles until tested, forty-eight hours after removal, did not contain a trace of chloroform vapor.

Mr. Duroy has employed a similar process for separating chloroform from the blood and viscera, but under a different arrangement. The flask is furnished with two tubes: through one, air can be propelled by a pair of bellows, and through the other the air charged with chloroform vapor is forced through a porcelain tube strongly heated by passing through a furnace. The porcelain tube is fitted at the other end with a Liebig's bulb-tube, containing a solution of nitrate of silver acidulated with nitric acid. The chloroform-vapor decomposed in the porcelain tube produces chloride of silver in the solution of the nitrate. This, it is assumed, is entirely derived from the decomposed chloroform vapor, and by weighing the precipitated chloride, the quantity of chloroform present may be determined. (Tardieu, *Sur l'Empoisonnement*, p. 844.) Any trace of volatile chloride might, however, give a similar result. This process fails to show the distinct presence of chlorine or hydrochloric acid in the decomposed vapor.

As chloroform is much more volatile than ether, and its odor is not so pungent, it is not so easily detected in the dead body by the smell. The body should be inspected as soon as possible, and any solids or liquids intended for examination should be kept in well-closed glass vessels. If the smell can still be perceived in the blood or organs, the vapor may be easily detected by the method above described. Chloroform, if not eliminated or lost by its volatility, may have been converted in the blood into formic acid, and thus removed from the ordinary method of research.

Treatment.—In poisoning by *liquid* chloroform the stomach-pump and emetics should be resorted to. As death frequently takes place suddenly from a suspension of the action of the heart (syncope), the treatment to be pursued, after the withdrawal of the chloroform vapor, consists in a free exposure of the face to a current of air, cold affusion, compression of the chest, and the maintenance of respiration by artificial means. The employment of stimuli, externally or internally, can be of little benefit, as sensibility is completely paralyzed by the poison. There can be no antidote to a poison which is diffused through the whole of the blood. The source of elimination is through the lungs; hence, as in poisoning by ether vapor, the only chance of restoration is in maintaining the act of breathing. The respiration of oxygen is not likely to be of practical benefit. The patient will either have recovered or died before this can be used. In cases of an asphyxial kind, in which the heart continues to beat after respiration has ceased, there is great hope of recovery by resorting to artificial respiration. (*Med. Times and Gaz.*, April 7th, 1858, p. 416; also June 26th, p. 662.)

Complete inversion of the body has been recommended as a method of treatment for restoring the cerebral circulation. (*Brit. Med. Journal*, August 22d, 1874, p. 237.) The lifeless condition, however, is probably owing as much to the altered state of the blood as to a deficient supply to the brain.

Dr. Hardie has published some cases showing the great benefit derivable from the application of the poles of an electro-magnetic battery to the sides of the neck. The current, conjoined with artificial respiration, restored the action of the lungs and heart in cases in which the patients appeared lifeless. He considers that death by chloroform vapor is the result of asphyxia, and not of any direct effect upon the heart. It is, in general, attributed to some "ill-defined diseased condition of the heart"—oftener supposed than discovered at the subsequent post-mortem examination. (*Lancet*, April 27th, 1872, p. 575.)

BICHLORIDE OF METHYLEN.

The vapor of this highly volatile liquid has been proposed by Dr. Richardson as a substitute for the vapor of chloroform in surgical operations. Mr. Miall, who has published a summary of one hundred cases, states that insensibility in adults was generally produced by the vapor in from two to five minutes, the average being three minutes and twenty seconds. It was thought to be less likely to cause death. Like all anæsthetic vapors, it has, however, destroyed life on several occasions, even when given with care. The history of these fatal cases is similar to that which chloroform vapor has furnished on numerous occasions.

Symptoms and Appearances.—In 1870 an operation for artificial pupil was about to be performed on a man, æt. 40. The vapor of the bichloride was given, and five minutes afterwards, when the operation had just commenced, the face of the man became livid, the breathing difficult, and the heart suddenly ceased to beat. On inspection the principal appearance was congestion of the lungs. In another case, which occurred in 1871, a man inhaled, for the purpose of a trivial operation, a drachm and a half of the bichloride. It was given in the usual way by an experienced person, and it was stated to be not more than one-half of the usual dose. The deceased became insensible—the operation was completed in a minute—when it was noticed that the patient's head had fallen on one side, his eyes were upturned, and breathing and pulsation had ceased. Animation could not be restored.

On inspection all the organs of the body were found healthy. There was no cause for death but the vapor of the bichloride (*Pharm. Journal*, 1871, p. 875). This preparation has been sometimes used in hospitals under the name of chloroform. In October, 1869, a man to whom the vapor was administered at Charing Cross Hospital, died in two minutes from the effects—although administered with care, and by one experienced in the use of chloroform. The allegation, therefore, that the vapor possesses any greater degree of safety than chloroform in surgical practice, is not supported by facts.

Analysis.—This liquid has a peculiar odor, resembling that of chloroform. It is not inflammable, but burns in contact with flame with a smoky combustion. It is not very soluble in water, but sinks in it, the globules having an opaque appearance. It has no acid reaction. Nitrate of silver gives no precipitate with it. In contact with sodium and a small quantity of water, it is rapidly decomposed without combustion; the liquid acquires a yellowish color, and chlorine is then readily detected in it by nitrate of silver.

A mixture of chloroform and ether has been sold as bichloride of methylen. On shaking this mixture with water the chloroform is separated and sinks.

AMYLEN.

The vapor of this liquid was introduced by the late Dr. Snow as a substitute for the vapor of chloroform. It produces a loss of sensibility, without causing complete coma or stupor. Its use has already led to at least two deaths, and it is not so safe an agent as chloroform vapor for surgical purposes. The only *appearance* met with in one fatal case was an emphysematous state of the lungs, or excessive dilatation of the air-cells (*Med. Times and Gazette*, April 4 and 18, 1857, pp. 332, 381); and in the other, a distension of the right cavities of the heart with dark fluid blood. There was no congestion of the brain, and no smell of amylen perceptible in the body. (*Med. Times and Gazette*, Aug. 8, 1857, p. 133.)

CHAPTER LXV.

NARCOTIC LIQUIDS AND VAPORS—SULPHIDE OF CARBON—COAL-NAPHTHA—PETROLEUM—BENZOLE—OIL OF TURPENTINE—WOOD-NAPHTHA OR SPIRIT—FUSEL OIL OR AMYLIC ALCOHOL—CAMPHOR—NITROBENZOLE—ANILIN—NITRO-GLYCERIN OR GLONOIN.

SULPHIDE OF CARBON.

THIS liquid is extensively used in the arts as a solvent for phosphorus, sulphur, caoutchouc, and iodine. Nothing is known of its effects on man as a liquid; and from its powerful and offensive odor it could not be readily administered with homicidal intention. The effects of its vapor have been chiefly observed among workers who employ the liquid. It produces headache, loss of appetite, colicky pains, impairment of vision and hearing, and causes general derangement of health, evidently by an operation on the nervous system. (See *Chem. News*, May 2, 1863, p. 216.) Several cases of poisoning by this vapor in vulcanized rubber factories have occurred to Dr. Bernhardt. (Husemann's *Jahresbericht*, 1872, p. 495.)

Analysis.—The odor and inflammability of this liquid are sufficient to identify it even in the smallest quantity.

COAL-NAPHTHA—PETROLEUM—BENZOLINE—KEROSENE.

The light oily product of the distillation of coal, known under the name of coal-napththa, and chemically described as a hydrocarbon, has caused death in one case, under symptoms of narcotic poisoning. A boy, æt. 12, swallowed inadvertently about three ounces of coal-naphtha, in the state in which it is used for burning in lamps. He soon appeared as if intoxicated, and ran about in a wild delirium. When seen in a short time by a medical man, he was insensible—in a state of collapse—breathing stertorously, and his skin was cold and clammy. He had already vomited part of the liquid, and the odor of the vomited matter at once showed that he had taken coal-naphtha. By the promotion of vomiting, he was made to eject altogether two tablespoonfuls of naphtha, and he partially recovered. In spite of this reaction, however, in about two hours he was again in a state of collapse, insensible, pulseless, gasping for breath, and frothing at the mouth. The eyes were fixed and glassy, and the pupils contracted. There was complete loss of muscular power, and great difficulty of breathing, but no convulsions. He had lost the power of swallowing. In spite of every effort to save him, he died in less than three hours after swallowing the liquid. On inspection of the body four days after death, a strong smell of naphtha was perceived throughout the tissues. The blood was fluid, there was a slight effusion of serum in the ventricles of the brain. The right side of the heart contained fluid blood, the left was empty, the lungs were not congested but pale. The coats of the stomach were not inflamed or materially changed in appearance. This organ contained a pint of semifluid matter, of which four or five ounces were liquid. An ounce of a dark-colored liquid floated on the top, and was easily skimmed off. This was found to be naphtha by its lightness, its insolubility in water, and by its inflammability. It burnt with a thick smoky flame. The liquid appeared to act in this case as a pure narcotic. There were no convulsions. The respiration of the *vapor* of this liquid diluted with air, produces headache, giddiness, severe pain in the stomach, loss of appetite, and general illness. (*Lancet*, Aug. 23, 1856, p. 230.)

Petroleum (*petræ oleum*) is a light mineral hydrocarbon oil found in the earth. As it is imported, in the form of rock oil, it contains *benzoline*, *kerosene*, etc. The effects of these liquids, as far as they are known, are similar to those of coal-naphtha.

A case is reported in which a woman, for the purpose of suicide, swallowed a pint of *petroleum*. There were slight symptoms of intoxication; slight pain in the stomach, and but little febrile excitement. There was a strong odor of petroleum about the body, and this smell was perceptible for six days. She entirely recovered. (*Amer. Jour. Med. Sci.*, April, 1873, p. 566.)

Analysis.—The peculiar odor as well as inflammability of these liquids, and the fact that they burn with a bright yellow smoky flame, would be sufficient to identify them. The lightness of coal-naphtha and its insolubility in water would, as in the case above mentioned, allow of its being rapidly separated from the aqueous contents of the stomach.

BENZOLE.

This is a colorless volatile liquid hydrocarbon obtained by the distillation and rectification of coal-naphtha. The breathing of its vapor produces narcotic effects, but with some symptoms indicative of a noxious action on the brain and spinal marrow, *e. g.*, noises in the head, convulsive trembling, twitchings of the muscles, convulsions, with difficulty of breathing. (See paper by Dr. Stone, *Med. Gaz.*, 1848, vol. 41, p. 1077.) But little is known concerning the action of liquid benzole on the human subject. It is poisonous to the lower animals and to all parasites; and it has been suggested by Dr. Sonnenkalb as a remedy for destroying the trichina spiralis (p. 521). This writer refers to a case in which a quantity of liquid benzole was swallowed by a man, and it operated as a narcotic. (*Anilin und Anilinfarben*, Leipzig, 1864, p. 13.)

Analysis.—The odor and inflammability of the liquid, as well as its insolubility in water, are sufficient to identify it, and allow of its separation from organic liquids.

OIL OF TURPENTINE—CAMPHINE.

The few cases in which this liquid has produced any noxious symptoms have occurred among children. From these it appears to have rather the effects of a neurotic (narcotic) than an irritant poison. In a dose of three drachms it has produced a kind of intoxication. A dose of a tablespoonful caused in a child, aged eighteen months, symptoms bearing a strong resemblance to those occasioned by an overdose of opium, although they were not so rapidly manifested. (See case by Mr. Johnson, *Med. Times*, Oct. 11, 1851, p. 380.) In three hours there was complete insensibility, with stertorous breathing, strongly contracted pupils, rapid and weak pulse, coldness of skin, paleness of the countenance, general relaxation of the muscles, and occasional convulsive movements. Two fatal cases are recorded. The first was that of a child, aged fourteen weeks. It occurred in January, 1869. I am indebted to Mr. Miall, of the Bradford Infirmary, for the particulars. The evidence at the inquest showed that the child had had half an ounce of the oil poured down his throat by a brother, æt. 8. He had been left asleep at 9 P.M., and in an hour was found to be insensible, cold, and slightly convulsed. At about 12 P.M. he was seen by Mr. Miall.

He was comatose, pale, with extremely cold surface; pupils contracted; slow and irregular breathing about three times in a minute, pulse quick, small, compressible, almost imperceptible. A strong odor of turpentine issued from the mouth, and there was a spot of liquid smelling of turpentine on the pillow. The child was unable to swallow. He died in fifteen hours after taking the poison. The second case occurred in Birkenhead in July, 1872. The child was five months old. A spoonful of spirit of turpentine was given to it by mistake for peppermint, and death took place rapidly. (*Pharm. Jour.*, July, 1872, p. 75.) Oil of turpentine is occasionally given medicinally to children suffering from worms. The above cases should inspire caution.

There is a case of recovery reported in which an infant had swallowed four ounces of the oil. Another case, in which the oil was criminally administered to an infant, was the subject of a trial at the Central Criminal Court, Dec. 1856 (*Reg.* v. *Rodanbosh*); it did not destroy life, but the child suffered for some time from the effects. The defence was that the oil of turpentine was poured down the child's throat by the mother with a view to cure it of a cough. She was acquitted.

Camphine is oil of turpentine simply purified by distillation with lime. A case of poisoning by this rectified oil occurred to Dr. Thomsen, of Schleswig. A woman, æt. 22, swallowed a large quantity of the oil. She was soon seized with violent vomiting, which was increased by milk and other liquids. The matter vomited smelt strongly of turpentine. She was restless, and in great pain; there was some purging. There was not an entire loss of consciousness. In two hours she complained of cold, the pulse was small and weak; the head then became hot, there was headache, but the pupils were unchanged; there was redness of the conjunctivæ. A quantity of urine was passed smelling of violets, and the breath also had a similar odor. There were some slight nervous symptoms, but these passed off. She recovered in eight days. (Horn's *Vierteljahrsschrift*, 1866, vol. 2, p. 337.)

WOOD-NAPHTHA—WOOD-SPIRIT—METHYL ALCOHOL.

The term naphtha is frequently applied to a product of the destructive distillation of wood, differing entirely in composition and properties from the hydrocarbon above described as coal naphtha. It is also known under the names of methyl alcohol and pyroligneous ether. It differs from coal-naphtha, among other properties, in being miscible with and soluble in water, in all proportions. It is a nauseous liquid in odor and taste. When mixed in the proportion of one-tenth part with rectified spirit, it forms a compound now largely employed as a solvent in the arts and medicine under the name of *Methylated spirit.* It has a hot disagreeable spirituous taste, and like rectified spirit it would no doubt operate as a narcotic poison. Its odor is so powerful and peculiar, that no one could swallow it unknowingly. I have met with only one instance of the effects of this liquid on man; but I have seen the effects produced by the respiration of its vapor on a large scale. It causes headache, loss of appetite, nausea, sickness, languor, and a general feeling of illness. A fatal case from an overdose of wood-spirit mixed with alcohol occurred in London in September, 1864, and was the subject of an inquest. The evidence showed, on an inspection of the body, that the lungs were congested and the stomach irritated and inflamed.

Analysis.—It is one of the most inflammable of liquids, burning with a pale blue flame. It is light and volatile, readily separable from other liquids by distillation below 200°. Its odor is peculiar. It mixes with water and alcohol in all proportions. Alcohol containing one-tenth part of it is rendered so nauseous that it is supposed to prevent the use of the mixture for the purposes of drinking.

FUSEL OIL—AMYLIC ALCOHOL.

This liquid is also known under the name of Potato-spirit or oil of grain. It is of an alcoholic nature, but much less volatile than alcohol and ether; hence it is commonly a product at the latter part of the distillation of spirit from fermented potatoes and cereal grains, imparting a disagreeable odor and taste to the brandies produced. Its vapor, when respired in a diluted state, is irritating to the lungs; it produces headache, nausea, and a feeling of giddiness, with a sense of suffocation and an inability to stand or walk. This spirit is used in certain manufactures, as in the separation of oils and fats, and a question has arisen how far the vapors would be injurious to the health of workmen. There can be no doubt that the vapor is noxious when breathed, and that the work could not be carried on with safety unless there were free and perfect ventilation.

Dr. Furst, of Berlin, found that two drachms of the liquid thrown into the stomach of a rabbit caused great restlessness and loss of muscular power; but the animal soon recovered. A similar quantity killed another rabbit in about two hours; the principal symptoms were great depression and difficulty of breathing. On inspection of the body, there was extravasation of dark-brown blood at the gullet end of the stomach, and the mucous membrane presented brownish-red points. The lining membrane of the upper part of the small intestines was reddened and covered with mucus; the kidneys were healthy and bloodless, and the lungs somewhat redder than natural. Three drachms killed a rabbit within an hour. Half an ounce caused death in a quarter of an hour, and one ounce in four minutes. Fusel oil, as a liquid, appears to have at first a stimulating and afterwards a depressing action. In small quantities it produces intoxication. (See *London Med. Gaz.*, vol. 35, p. 430.) This liquid is absorbed into the blood, and after a time may be detected by its peculiar odor in the breath. Its toxicological effects are more powerful in the state of vapor, than when it is taken as a liquid into the stomach.

Analysis.—Fusel oil is a volatile liquid of a pale yellow color, lighter than water and only sparingly soluble in it. It is dissolved by alcohol and ether in all proportions, but not readily by chloroform. Water separates it from its ethereal solution. It has a hot burning taste and an offensive spirituous odor, which is very persistent and peculiar; by this it may be distinguished from other alcoholic liquids. It is inflammable, and burns with a pale bluish flame. Like alcohol, ether, and wood-spirit, it decomposes chromic acid, producing green oxide of chromium. In organic mixtures ether might be used for its separation. By distilling fusel oil with acetate of potash and oil of vitriol, an ethereal liquid is produced, which is used in confectionery under the name of *Essence of Jargonelle Pear.*

CAMPHOR.

There are but few instances recorded in which camphor has proved fatal in the human subject; but it has on several occasions produced alarming symptoms, and would probably have destroyed life had it not been early removed from the stomach. In the few cases that have been observed, its effects were somewhat different.

Symptoms and Appearances.—Camphor operates on the brain and nervous system. A woman swallowed about *twenty grains* of camphor dissolved in rectified spirit of wine mixed with tincture of myrrh. In half an hour she was suddenly seized with languor, giddiness, partial loss of sight, delirium, numbness, tingling and coldness of the extremities, so that she could hardly walk. The pulse was quick, and breathing difficult, but she suffered no pain in any part. On the administration of an emetic, she vomited a yellowish liquid, smelling strongly of camphor. In the evening the symptoms were much diminished, but she had slight convulsive fits during the night. The next day she was convalescent; the difficulty of breathing, however, continued more or less for several weeks. This is the smallest dose of camphor which appears to have been attended with serious symptoms in an adult. A man, æt. 39, swallowed about *thirty-five grains* of powdered camphor, prepared for lozenges. In twenty minutes giddiness and dimness of sight came on, and he fell from a chair in a kind of epileptic fit, which lasted about ten minutes. The limbs were cold; the pulse was frequent and scarcely perceptible. When roused he had scarcely power to articulate. A quantity of a clear liquid, smelling strongly of camphor, was drawn off by the stomach-pump. The man did not recover for a week, suffering chiefly from general exhaustion and suppression of urine; this latter symptom continued more or less for three months afterwards. There was no disorder of the stomach or bowels. A man, who had eaten an unknown quantity of camphor, suffered from the following symptoms: A burning sensation in the stomach; a painful feeling in the head, as if it were pressed in a vice. There was restlessness; vertigo; a desire to walk about; he staggered like a drunken man, and became completely insensible. His limbs were cold; his face was pale; his body convulsed, and the pupils were dilated. Emetics were given, and he soon afterwards became sensible. During the next day there was a great desire to sleep; he slept twenty-four hours, and after two or three days was convalescent. (*Med. Times and Gaz.*, 1858, vol. 2, p. 645.)

Dr. G. Johnson has recently directed the attention of the profession to the noxious effects of the homœopathic solution of camphor, which is a saturated solution of this substance in alcohol (one ounce of camphor dissolved in one ounce and a quarter of alcohol). It is, therefore, much stronger than the *Spiritus Camphoræ*, B. P., and may be mistaken for it. Dr. Johnson describes the effects of comparatively small doses in three cases. Case 1. A lady, æt. 20, took for a cold and sore throat twenty-five drops of the homœopathic solution of camphor in water. She went to bed, and in a short time was found foaming at the mouth, black in the face, and violently convulsed. In spite of medical treatment, she was unconscious for several hours. She vomited a fluid tinged with blood, and smelling strongly of camphor, and had severe pain in the stomach. For several days she was partially paralyzed, and six months afterwards she was still suffering from symptoms of nervous derangement. Case 2. A clergyman took three drops of the same solution every five minutes for an hour. After taking the eighth dose, he was seized with intense headache, which confined him to bed for forty-eight hours, and he was afterwards so weak and ill that he was unable to enter his pulpit for two months. Case 3. A young lady, æt. 19, took for diarrhœa a teaspoonful of the same preparation, which rendered her comatose for several days, and caused a variety of nervous symptoms, continuing for some days longer. In another case, seven drops of the solution on a lump of sugar produced alarming symptoms. In the *British Medical Journal* for Feb. 27, 1875, p. 272, Dr. Johnson describes the case of a strong healthy Eton boy, æt. 14, who took as a remedy for a cold fifteen drops of *Rubini's Homœopathic Camphor*. Soon afterwards he was found lying on his bed insensible, almost pulseless, with cold extremities, and his face and lips were pallid. An emetic brought the noxious drug off the stomach, and he recovered. He stated that after swallowing the camphor on sugar he became insensible. Dr. Johnson states that this is the seventh case of poisoning with homœopathic camphor which has come to his knowledge within the last two years. Cases of a similar kind have fallen under the notice of other observers. (*Brit. Med. Jour.*, Feb. 1875.)

It has been objected to Dr. G. Johnson's conclusions respecting the poisonous effects of camphor, that larger doses than he describes as poisonous have been taken, not only with impunity, but with benefit; but the objectors have overlooked the fact that poisoning depends on the facility with which a substance is absorbed. Camphor, taken as a solid in powder, would not produce the same results as when taken in the form of a saturated solution in alcohol or chloroform.

In January, 1863, an infant of fifteen months died from the effects of some camphorated oil given to it by mistake. Convulsions ensued, and death took place in thirteen hours. Three cases of poisoning by camphor are reported by Dr. Schaaf, one of which proved fatal. A woman gave about thirty grains (half a teaspoonful) of powdered camphor to each of her three children as a vermifuge. Two of the children were respectively of the ages of three and five years; the third was an infant aged eighteen months. The first symptoms were paleness of the face, with a fixed and stupid look. Delirium followed, with a sense of burning in the throat, and great thirst. Vomiting, purging, and convulsions supervened, and in one child the convulsions were most violent. The two elder children, after suffering thus for three hours, flammable, and burns with a pale bluish flame. Like alcohol, ether, and wood-spirit, it decomposes chromic acid, producing green oxide of chromium. In organic mixtures ether might be used for its separation. By distilling fusel oil with acetate of potash and oil of vitriol, an ethereal liquid is produced, which is used in confectionery under the name of *Essence of Jargonelle Pear.*

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Dr. G. Johnson has recently directed the attention of the profession to the noxious effects of the homœopathic solution of camphor, which is a saturated solution of this substance in alcohol (one ounce of camphor dissolved in one ounce and a quarter of alcohol). It is, therefore, much stronger than the *Spiritus Camphoræ*, B. P., and may be mistaken for it. Dr. Johnson describes the effects of comparatively small doses in three cases. Case 1. A lady, æt. 20, took for a cold and sore throat twenty-five drops of the homœopathic solution of camphor in water. She went to bed, and in a short time was found foaming at the mouth, black in the face, and violently convulsed. In spite of medical treatment, she was unconscious for several hours. She vomited a fluid tinged with blood, and smelling strongly of camphor, and had severe pain in the stomach. For several days she was partially paralyzed, and six months afterwards she was still suffering from symptoms of nervous derangement. Case 2. A clergyman took three drops of the same solution every five minutes for an hour. After taking the eighth dose, he was seized with intense headache, which confined him to bed for forty-eight hours, and he was afterwards so weak and ill that he was unable to enter his pulpit for two months. Case 3. A young lady, æt. 19, took for diarrhœa a teaspoonful of the same preparation, which rendered her comatose for several days, and caused a variety of nervous symptoms, continuing for some days longer. In another case, seven drops of the solution on a lump of sugar produced alarming symptoms. In the *British Medical Journal* for Feb. 27, 1875, p. 272, Dr. Johnson describes the case of a strong healthy Eton boy, æt. 14, who took as a remedy for a cold fifteen drops of *Rubini's Homœopathic Camphor*. Soon afterwards he was found lying on his bed insensible, almost pulseless, with cold extremities, and his face and lips were pallid. An emetic brought the noxious drug off the stomach, and he recovered. He stated that after swallowing the camphor on sugar he became insensible. Dr. Johnson states that this is the seventh case of poisoning with homœopathic camphor which has come to his knowledge within the last two years. Cases of a similar kind have fallen under the notice of other observers. (*Brit. Med. Jour.*, Feb. 1875.)

It has been objected to Dr. G. Johnson's conclusions respecting the poisonous effects of camphor, that larger doses than he describes as poisonous have been taken, not only with impunity, but with benefit; but the objectors have overlooked the fact that poisoning depends on the facility with which a substance is absorbed. Camphor, taken as a solid in powder, would not produce the same results as when taken in the form of a saturated solution in alcohol or chloroform.

In January, 1863, an infant of fifteen months died from the effects of some camphorated oil given to it by mistake. Convulsions ensued, and death took place in thirteen hours. Three cases of poisoning by camphor are reported by Dr. Schaaf, one of which proved fatal. A woman gave about thirty grains (half a teaspoonful) of powdered camphor to each of her three children as a vermifuge. Two of the children were respectively of the ages of three and five years; the third was an infant aged eighteen months. The first symptoms were paleness of the face, with a fixed and stupid look. Delirium followed, with a sense of burning in the throat, and great thirst. Vomiting, purging, and convulsions supervened, and in one child the convulsions were most violent. The two elder children, after suffering thus for three hours, fell into a comatose sleep, and on awaking, the symptoms passed off. The infant died in seven hours, not having manifested any return of consciousness from the first occurrence of convulsions. (*Journal de Chimie Médicale*, 1850, p. 507.) The severity of the symptoms is fully explained by the large quantity administered and the age of the children. In a dose of one drachm given in a clyster, camphor produced alarming symptoms. (*Med. Gaz.*, vol. 48, p. 552.) In a case reported in the *Medical Gazette* (vol. 11, p. 772), 120 grains were taken by a physician, and all that he experienced was lightness in the head with great exhilaration. There was no derangement of the stomach or bowels. He slept profoundly for some hours, and awoke very weak and exhausted. He also perspired greatly during his sleep. It is difficult to draw any conclusion from this case, as the quantity taken was conjectural, and the patient, while laboring under the effects of the poison, was not seen by any person.

A soldier took a large quantity of camphor daily. For three days it had no effect upon him. On the fifth day he suffered from great pain and a burning sensation in the stomach. His head was painful; there was giddiness, with an incessant desire to walk about, although like a drunken man, he could hardly keep on his legs. He soon fell completely insensible; his limbs were cold, his face was pale, his body convulsed, and the pupils were dilated. These symptoms were followed by an irresistible desire to sleep. In two or three days he recovered. (*Med. Times and Gaz.*, Dec. 1858, p. 645.)

M. Raspail has advocated the use of camphor in large doses as a universal remedy for diseases. This rash practice has been in some instances attended with dangerous effects. A man who had taken about sixteen grains in divided doses in twenty-four hours, complained of a sense of suffocation, difficulty of breathing, sickness, and great anxiety. (*Jour. de Pharmacie*, Fév., 1846, p. 121.) In the same journal three other cases are mentioned, in which alarming effects followed the injudicious use of this drug. The largest dose of camphor that has been taken was in a case which occurred to Wendt, of Breslau. Eight scruples (160 grains) were swallowed by a drunkard, dissolved in spirit. The symptoms were giddiness, dimness of sight, delirium, and burning pain in the stomach. There was *no vomiting;* the man recovered! (Wibmer, *op. cit.*, vol. 3, p. 212.) In Orfila's experiments on animals, the mucous membrane of the stomach was found inflamed (vol. 2, p. 493).

Treatment.—The free use of emetics and stimulants.

Analysis.—Camphor would probably be found in the state of lumps, or dissolved in spirit. No difficulty will occur in identifying this substance when it has proved fatal and is found in the contents of the stomach. Its presence would be immediately recognized by its powerful and peculiar odor, which has been perceived throughout the whole body in dogs poisoned with it. If it were diffused in the form of lumps or powder, these might be easily separated from the contents, owing to the great insolubility of this substance. In general, it might be expected that some portions would float on the surface of water, in which it is very insoluble. In a doubtful case, the solid contents of the stomach may be concentrated and treated with a large quantity of alcohol, the alcoholic liquid filtered, and the camphor separated by adding water. It is a white solid, possessing a well-known odor, easily dissolved by alcohol, and again separated by water, entirely volatile without residue, and burning with a rich yellow smoky flame. Camphor is soluble in chloroform, and this liquid may be used as a means of separating it from organic liquids.

NITROBENZOLE.

This liquid, which is largely employed as a substitute for the essential oil of bitter almonds in perfumery and confectionery, has now taken its place among narcotic poisons. In the second edition of this work (*On Poisons*, 1859) some experiments were quoted from the *Lancet*, (Jan. 10, 1857, p. 46), showing that one drachm of nitrobenzole killed a rabbit almost instantaneously; and half a drachm, mixed with two drachms of water, rendered a cat insensible for several minutes, a slimy mucus flowing from its mouth for several hours afterwards. The animal refused all food, and died in twenty-four hours. In 1859, the late Professor Casper, of Berlin, published an account of this liquid under the name of "A new Poison." (*Vierteljahrsschrift*, B. 16, p. 1.) Its effects on a rabbit and a dog are here described. Two drachms of it were given to a rabbit without any symptoms being produced; two drachms were then given to the animal at intervals of ten minutes or a quarter of an hour, until the animal had taken one ounce. In a minute and a half after the last dose, the animal fell suddenly on its left side. The pupils were dilated, while the limbs and tail were strongly convulsed. The animal died in another minute. The dose was probably unnecessarily large, but the result shows that nitrobenzole in a large dose destroys life rapidly. On opening the body, the powerful odor of the liquid was everywhere perceptible, even in the blood. This odor remained strongly in the body when it was again examined fourteen days after death. Twenty cubic centimetres (about five drachms) given to a middle-sized dog produced no remarkable symptoms. After some hours the animal was observed to be dull and languid; in twelve hours there was profound coma, with slow respiration and coldness of the skin; but there were no convulsions. The animal was then killed. All the solids and liquids of the body, including the blood, had a strong odor of the poison; and some drops of the oily liquid were separated from the contents of the stomach. The fluid on which it floated had a strong alkaline reaction. The blood retained the odor for several days.

Symptoms.—Passing from experiments on animals to the effects produced on man, the cases hitherto observed show that this is a most insidious poison, both in liquid and vapor, and, as in the action of chloroform and fusel oil, the vapor is much more potent than the liquid. There is a burning taste in the mouth, followed by a sensation of numbness and tingling in the tongue and lips. There is no immediate insensibility, as in poisoning by prussic acid, and there are no convulsions. The eyes are bright and glassy, the features pale and ghastly, the lips and nails purple, the skin clammy, and the pulse feeble. There is a powerful odor resembling that of oil of bitter almonds. The mind may be clear for an hour or several hours after the poison has been swallowed. The patient then becomes suddenly unconscious, the jaws fixed, the hands clenched and blue, and the muscles rigid and convulsed. In one case there was vomiting of a liquid having the odor of nitrobenzole. The breathing was slow, and the pulse scarcely perceptible. Reaction set in, in about eleven hours, and recovery took place. (*Guy's Hospital Reports*, Oct. 1863, p. 192.) Mr. Nicholson (*Lancet*, Feb. 1, 1862, p. 135), in referring to a fatal case of poisoning by the liquid, states that he has known several instances in which the *vapor*, as it is evolved from almond glycerin soap, has seriously affected persons. A friend of his who used a cake of the soap in taking a warm bath fainted from the effects of the vapor of nitrobenzole set free, and was ill for some time afterwards.

In July, 1863, Mr. Fotherby communicated to me a case of poisoning by this compound, in which the symptoms so closely resembled those of essential oil of bitter almonds, that it was at first supposed this oil had been taken. A woman, æt. 30, tasted a liquid which had been used for flavoring pastry, and perceiving that it was very acrid on her tongue and lips, spat it out immediately and washed her mouth with water. She thought she could not have swallowed more than a drop, but in replacing the bottle she spilled about a tablespoonful on the table and did not immediately wipe it up. The vapor strongly impregnated the small room in which she was, and produced a feeling of sickness in another servant. The burning taste in the mouth was immediately followed by a sensation of numbness and tingling in the tongue and lips, and a strange feeling for the next hour. As the woman became worse, Mr. Fotherby was called in, and saw her in an hour and three-quarters after the occurrence. Her aspect was then quite typical of prussic acid poisoning: the eyes were bright and glassy, the features pale and ghastly, the lips and nails purple, as if stained with blackberries; the skin was clammy, and the pulse feeble. Her mind was then clear, and she described how the accident had occurred and what her sensations were. She was able to swallow a mustard emetic, after which she became rapidly worse, lost her consciousness, the teeth were set, the hands were clenched and blue, the muscles rigid and convulsed. She vomited freely a pale fluid matter, which had the peculiar odor of nitrobenzole. The stomach-pump was used, but the fluid washed out of the organ had hardly any odor, owing probably to the small quantity actually swallowed, and its removal by absorption. The breathing was much reduced, and the pulse could scarcely be felt. In about eleven hours there was reaction, consciousness returned, and she was able to swallow. At the end of seventeen hours she was much better; but she then complained of distorted vision, with flashes of light and strange colors before her eyes. For some weeks she continued weak. It was at first supposed the woman had swallowed a larger quantity of the liquid than she had imagined; but it is obvious from the entire absence of the odor in the fluid drawn off by the stomach-pump, within about two hours, that but little could have passed into the stomach. There is no doubt, from what has been observed in other cases, that these severe symptoms were chiefly due to the breathing of the vapor in a concentrated form. A fellow-servant who was in the room at the time that the nitrobenzole was spilled, also suffered from the inhalation of the vapor. Mr. Fotherby sent me a portion of the liquid, and I found it to be pure nitrobenzole unmixed with any essential oil of almonds.

A case of poisoning with this liquid, which was the subject of an inquest at Ramsey, in the Isle of Man, is reported in the *Pharmaceutical Journal*, for December, 1862, p. 283. A clerk in some chemical works took, on the 6th of November, a few drops (supposed to have been fifteen) of nitrobenzole. Immediately afterwards he felt unwell and became insensible. Stimulants restored consciousness, but there was a relapse, and he died the next day. The following case occurred at the London Hospital: A boy, æt. 17, while drawing off some nitrobenzole by a siphon, swallowed a portion of the liquid. There were no immediate symptoms, but he soon felt sleepy, and when at dinner, ate but little, and said he felt as if he was drunk. This was between two and three hours after he had swallowed the liquid. He fell into a stupor, which became deeper and deeper until death took place, without vomiting or convulsions, twelve hours after the ingestion of the poison. (Dr. Mackenzie, in *Med. Times and Gazette*, 1862, vol. 1, p. 239.) The two following cases occurred at Maidstone, in April, 1865. A boy, æt. 13, applied a bottle containing nitrobenzole to his lips. No symptoms followed at the time, and the boy ate his dinner as usual. Some hours elapsed, when he suddenly became insensible. He was almost pulseless; his jaws were spasmodically closed; the skin of his face was purple, and his lips were livid. He died in about four hours after the seizure, and twelve hours after taking the poison. Some small quantity was most probably swallowed, as the contents of the stomach had a strong smell of the liquid. A cook in the same family also applied the bottle to her lips. It tasted bitter. She had her dinner as usual, but an hour after tasting the nitrobenzole, she was seized with vomiting and felt very ill; her lips were black, and her face was purple and white. The woman recovered. The poison had been wrongly labelled oil of bitter almonds. For several cases of poisoning by this liquid, see Husemann's *Jahresbericht*, 1872, p. 531; and a paper by Dr. Schenk, Horn's *Vierteljahrsschrift*, 1866, vol. 1, p. 32.

In a paper communicated to the Royal Society in 1863, Dr. Letheby describes two cases which fell under his observation. In one, a man, æt. 43, spilled a quantity of nitrobenzole over his clothes, and went about several hours, breathing an atmosphere saturated with the vapor. The effects were nearly the same in both cases, although in one the poison was inhaled in vapor, and in the other it was swallowed as a liquid. For some time there was no feeling of drowsiness in the man; gradually, however, his face became flushed, his expression stupid, and his gait unsteady; he had the appearance of a person who had been drinking. The stupor gradually increased, until it passed into profound coma, and in this state he died. The progress of each of the fatal cases was much the same as that of slow intoxication, excepting that the mind was perfectly clear, until the coming on of the fatal coma. This was sudden, like a fit of apoplexy; and from that moment there was no return of consciousness or bodily power; the sufferer lay as if in deep sleep, and died without a struggle. The duration of each case was nearly the same. About four hours elapsed from the time of taking or inhaling the poison to the setting in of the coma, and the coma lasted five hours.

Appearances after Death.—In these two cases, which may be taken as types of this form of poisoning, the following appearances were met with: a flushed face and livid lips; the superficial vessels of the body, especially about the throat and arms, were gorged with blood, which was everywhere black and fluid. The dependent parts were turgid; the lungs somewhat congested; the cavities of the heart were full of blood; the liver was of a purple color, and the gall-bladder distended with bile; the brain and its membranes were congested, and in one case, there was much bloody serosity in the ventricles. Nitrobenzole, as well as anilin, into which it appears to be partially converted in the body, was detected in the brain and stomach. (*Proc. Royal Soc.*, 1863, No. 56, p. 550.) It is not stated what the result of the analysis, if any, was in reference to the case of death from the vapor, in which the poison was absorbed through the lungs. In performing some experiments on animals, Dr. Letheby found that the local action on the stomach was slight; there was rarely any vomiting, and there was either rapid coma, or a slow setting in of paralysis and coma, after a long period of inaction. There was a complete loss of voluntary power, a spasmodic fixing of the muscles of the back, with violent struggles, a look of distress, and occasionally a kind of epileptic fit. The pupils were widely dilated, the action of the heart was irregular, and the breathing difficult. The time of death in more rapid cases varied from twenty-five minutes to twelve hours after the administration of the poison. In other experiments, in which smaller doses were given, the time that elapsed between the administration of the poison and the coming on of the first symptoms (an epileptic fit) varied from nineteen to seventy-two hours; in most cases it was about two days, and the time of death was from four to nine days. The appearances were similar to those already described. When death had taken place within twenty-four hours, the odor of the nitrobenzole was clearly perceptible in the stomach, brain, and lungs; and anilin (from the chemical conversion of nitrobenzole) was found in the organs. In the slower fatal cases the odor had often entirely disappeared; but traces of anilin could be detected in the brain and urine, and sometimes in the stomach and liver. Occasionally no trace of the substance was found, although death had taken place from the poison.

This narcotic compound differs from the ordinary narcotics in its powerful and persistent odor, which would render it difficult for a person to administer it, either in liquid or vapor, unknowingly to another; in the production of profound coma at an uncertain interval after the stupor; and in the rapidly fatal effects when coma has followed. It operates powerfully as a poison in vapor as well as in a liquid state; but so far as cases have yet been observed in the human subject, the symptoms resembling those of the first stage of narcotic poisoning have very soon appeared. The rapidly fatal cases only, would be likely to be mistaken for apoplexy, but in these the poison would be detected by its odor.

Analysis.—Nitrobenzole or *Essence of Mirbane* is a pale lemon-colored oily liquid of a strong odor resembling that of bitter almonds. It has a pungent hot disagreeable taste. It gives to confectionery the smell but not the pleasant taste of the oil of bitter almonds. It destroys the color of litmus, and gives a greasy stain to paper, leaving a yellow mark when the stain disappears. It sinks in water, and is partially dissolved, giving to it a yellowish color. It is soluble in alcohol, ether, and chloroform; but when agitated with water, it is in great part separated from its ethereal and chloroformic solutions. It has no basic qualities; its aqueous solution is not precipitated either by tannic acid or the chloriodide of mercury and potassium. It is highly combustible, burning with a yellow smoky flame. It yields no Prussian blue when mixed with sulphate of iron, alcohol, and potash, and its vapor produces no cyanide of silver with a solution of the nitrate. It is distinguished from all other liquids, excepting the essential oil of almonds, by its odor, and from this oil by the following tests: Pour a few drops of each on a plate and add a drop of strong sulphuric acid. The oil of almonds acquires a rich crimson color with a yellow border, the nitrobenzole produces no color. In order to separate it from organic liquids, they may be acidulated with sulphuric acid, and submitted to distillation in an apparatus similar to that which is described at page 320, Fig. 22. If any of it exists in a free state, its odor will be sufficient for detecting its presence. If converted into anilin, another process will be required. There is no probability that this liquid will be successfully employed for the purposes of murder without the certainty of detection.

ANILIN.

This is a narcotic poison in liquid or vapor, resembling nitrobenzole in its toxicological effects. Schuchardt found that a small rabbit was killed by sixty drops in six hours and a quarter, and a large rabbit by one hundred drops in four hours. There was loss of sensibility with loss of heat, and violent clonic and tonic convulsions ensued which continued until death. From experiments hitherto performed, it does not appear to be an active poison as a liquid, and it seems to affect the spinal marrow more than the brain. It has also a local irritant action. Dr. Turnbull gave half a drachm of the sulphate to a dog. In two hours and a half the animal vomited, and an hour later it was purged. It became dull, weak, and tremulous; the pulse was rapid, and the breathing labored. The feet were cold, and the hind legs paralyzed, and the tongue was of a blue color. In five hours the symptoms abated, and the next day the animal had recovered. (*Lancet,* Nov. 16, 1861.)

Dr. Letheby found that anilin given to dogs and cats in doses of from twenty to sixty drops, caused a rapid loss of voluntary power. The animal staggered and fell upon its side powerless, the head was drawn back, the pupils were dilated, the breathing was difficult, and the action of the heart tumultuous; there were slight twitchings or spasms of the muscles, and the animal quickly passed into a state of coma, from which it did not recover, death taking place in from half an hour to thirty-two hours. On inspection the brain and its membranes were congested, the cavities of the heart were nearly full of blood, and the lungs but slightly congested. The blood all over the body was black and coagulated. The poison was easily discovered in the brain, the stomach, and the liver, although it was found that, as nitrobenzole is changed into anilin, so in some cases anilin and its salts are converted into mauve and magenta. This arises from the oxidation of the salts, and it has been especially observed on the surface of the body. (*Proc. Royal Society*, No. 56, 1863, p. 556.) I have not met with any instance recorded of the effects of anilin as a poison on the human body. It is usually seen as a dark-colored liquid of nauseous odor and taste, and it could hardly be taken or administered without the consciousness of the person taking it. The salts appear to have very little action. They have been used medicinally in large doses without producing any unusual effects. In one case 406 grains of the sulphate of anilin were given to a patient in the London Hospital, in the course of a few days, without any symptoms of poisoning. (Dr. Letheby, loc. cit. See also cases by Dr. Fraser, *Med. Times and Gazette*, March 8, 1862, p. 239.) It is difficult to suppose that combination with an acid to form a perfectly soluble salt can render anilin inert, as this would be contrary to experience in reference to other bases, *e. g.*, nicotina and conia; at the same time, if we except the action of the vapor, no case of poisoning has occurred which will enable us to solve this question.

There are facts which show that the *vapor* of anilin, even when much diluted, exerts a noxious effect on man. Mr. Knaggs met with a case in which a workman accidentally broke a carboy containing a large quantity of this liquid; the anilin fell over him, but none entered his mouth. In his anxiety to wipe up the anilin, he respired the vapor for some time, felt giddy, and complained of his head and chest. When seen some hours after the accident his face and body were of a livid leaden hue, the lips, gums, tongue, and eyes of a corpse-like bluish pallor; he was breathing by gasping, and appeared at the point of death. There were no convulsions; he was sensible, and able to give a correct account of his feelings. His pulse was small and irregular. Under active treatment he recovered. (*Pharm. Jour.*, July, 1862, p. 42.)

Dr. Letheby met with the following case: In June, 1861, a boy, æt. 16, was brought into the London Hospital in a semi-comatose condition. In scrubbing out an anilin vat he had breathed the *vapor;* and although he did not suffer pain or discomfort at the time, he was suddenly seized with giddiness and insensibility. When brought to the hospital he looked like a person in the last stage of intoxication; the face and surface of the body were cold, and the pulse was slow and almost imperceptible, the action of the heart was feeble, and the breathing heavy and laborious. After rallying a little, he complained of pain in his head and giddiness. His face had a purple hue, and his lips, the lining membrane of his mouth, as well as his nails, had a similar purple tint. On the next day the narcotic symptoms had passed away, but he was remarkably blue, and looked like a patient in the last stage of Asiatic cholera. These cases appear to show that anilin vapor is less poisonous than that of nitrobenzole, and that the symptoms follow more rapidly on the inhalation of the vapor. Dr. Kreuser, of Stuttgart, has noticed among the workers in anilin that they have suffered from intense bronchitis, with a violent dry spasmodic cough, accompanied by ulcerations on the scrotum and extremities. The parts were swollen and painful, and covered with thick black crusts. This was obviously from want of cleanliness. (*Ed. Monthly Jour.*, Aug. 1864, p. 172.) For a full account of the effects of anilin on animals, I must refer the reader to a pamphlet by Dr. Sonnenkalb, of Leipsic, *Anilin und Anilinfärben in Toxikologischer und medicinal-polizeilicher Beziehung*, Leipzig, 1864, p. 20. The injurious effects to public health likely to arise from the employment of anilin colors in confectionery and cosmetics, are also fully described in this essay. Some of the anilin dyes by contact with the skin have produced much irritation and sometimes an eczematous state. This subject has attracted much attention in Germany. (See Eulenberg's *Vierteljahrsschrift*, 1871, vol. 2, p. 325.) Many mineral substances of an irritant and poisonous nature are used in the preparation of these dyes, and the dyed articles are not always freed from them by washing. (*Ann. d'Hyg.*, 1874, p. 371.)

Analysis.—Commercial anilin is an oily liquid of a reddish-brown color, with a peculiar tarry odor. It produces a greasy volatile stain on paper. It is volatile and combustible, burning with a thick smoky flame. It falls to the bottom of water, and does not readily dissolve in it. It is quite soluble in alcohol and ether, but not in chloroform; in the latter property it differs from nitrobenzole. Diluted sulphuric acid combines with it to produce a white compound which is dissolved by water. A solution of chloride of lime added to the acid watery liquid produces a splendid color, of various shades of purple and red.

The solution of sulphate of anilin is not precipitated either by tannic acid or chloriodide of mercury and potassium; but anilin itself, in the small quantity in which it is dissolved by water, yields, like the alkalies, a yellow precipitate with arsenio-nitrate of silver. It also reduces completely a solution of chloride of gold, precipitating metallic gold. A minute quantity of anilin may be thus detected. When pure anilin is heated with powdered corrosive sublimate, it produces a rich crimson dye. When present in organic liquids, anilin may be separated by digesting the concentrated liquid in alcohol, mixed with a little diluted sulphuric acid. The alcoholic extract, distilled at a high temperature with a solution of potash, yields anilin in the receiver. This may be tested by the methods above described.

NITROGLYCERIN (GLONOIN).

This is a powerfully explosive liquid, well known to chemists as a substitution-compound of the innoxious liquid glycerin obtained in the process of saponification. It has a sweet aromatic pungent taste, and it is stated that a single drop placed on the tongue produces a painful aching in the back of the head, which lasts for some hours. (Miller's *Chemistry*, vol. 3, p. 277.) Mr. Field states that he found one drop of the liquid, dissolved in water, produced insensibility and other symptoms of narcotic poisoning. (*Chem. News*, Nov. 7, 1863.)

Symptoms and Appearances.—In Sweden this liquid is much used in mining under the name of "blasting oil." Within the last four years ten cases of poisoning by it have occurred in that country. In these cases, the oil appears to have been taken in quantity (some ounces). In some instances it proved rapidly fatal. In a recent case a miner swallowed two mouthfuls. A painful feeling in his throat made him aware of his mistake, and he drank a quantity of milk. He was not seen by a medical man for an hour and a quarter. He was then suffering from faintness, difficulty of breathing, and oppression at the chest. In five hours vomiting and purging set in. Shortly before death the lips were livid, and the man lay quietly as if asleep, breathing feebly and occasionally with a deep sigh. On inspection, there was great congestion of the membranes of the brain as well as of the lower lobes of the lungs. The mucous membrane of the air-passages was of a red-brown color. The greater end of the stomach presented a similar appearance, with some ecchymosis. (Husemann's *Jahresbericht*, 1872, p. 533.)

According to Mr. Merrick, the vapor of this liquid acts powerfully as a narcotic poison, and even when much diluted with air it produces intense headache. Other experimentalists have not observed these extraordinary effects, and are inclined to regard it as inert, or at any rate they consider that its narcotic properties have been greatly exaggerated. Some support has been given to this last opinion by the fact that the liquid has been used by homœopathists under the name of glonoin, and the effects said to have been produced by infinitesimal doses are of so marvellous a character as to justify utter incredulity. Like other liquids described in this chapter, it probably acts most powerfully by its vapor, but further observations of a trustworthy kind are required to determine its potency as a poison.

Professor Vrij, of Rotterdam, has prepared nitroglycerin in large quantities, and has examined its chemical and physiological properties. Sobrero, who discovered it in 1847, stated that the smallest quantity was sufficient to produce the most violent headache, and he concluded from this that it was a powerful poison. Professor Vrij found that the vapor caused intense headache, but that it had no poisonous properties. He gave two drops to a rabbit, and no symptoms of poisoning were produced. (*Pharm. Journal*, 1855–6, p. 229.)

Analysis.—Nitroglycerin is a heavy oil-looking liquid. It is dissolved by water, but is insoluble in alcohol or ether. It explodes violently when struck or subjected to concussion.

CHAPTER LXVI.

POISONING WITH HENBANE—SYMPTOMS AND EFFECTS—HYOSCYAMIA—LACTUCARIUM—LACTUCIN—SOLANUM—SOLANIA—COCCULUS INDICUS—PICROTOXIN—INDIAN HEMP—CANNABIS.

HENBANE (HYOSCYAMUS NIGER).

ALL the parts of this plant are poisonous. The seeds produce the most powerful effects, then the roots, and lastly the leaves. The vapor evolved from the fresh-cut leaves has been known to produce giddiness, stupor, and syncope. In small or medicinal doses, henbane has a narcotic action; but when taken in large doses, it produces effects on the spinal marrow as well as on the brain.

Symptoms.—The best summary of these is given by Wibmer (*Arzneimittel*, art. " Hyoscyamus niger"). When the dose is not sufficient to destroy life, the symptoms are, general excitement, fulness of the pulse, flushing of the face, weight in the head, giddiness, loss of power and tremulous motion of the limbs, somnolency, dilatation of the pupils, double vision, nausea, and vomiting. After a time these symptoms pass off, leaving the patient merely languid. When a large quantity of the *root* or *leaves* has been eaten, an accident which has occurred from the plant having been mistaken for other vegetables, more serious effects are manifested. In addition to the above symptoms, in an aggravated form, there will be loss or incoherency of speech, delirium, confusion of thought, insensibility, coma, and, sometimes, a state resembling insanity; the pupils are dilated and insensible to light, there is coldness of the surface, cold perspiration, loss of power in the legs, alternating with tetanic rigidity and convulsive movements of the muscles; the pulse small, frequent, and irregular, the respiration deep and laborious. (See *Med. Gaz.*, vol. 47, p. 640.) Occasionally there is nausea, with vomiting and purging. Death takes place in a few hours or days, according to the severity of the symptoms. The special effect of this poisonous plant is manifested in its tendency to produce a general paralysis of the nervous system.

As an instance of the singular train of symptoms occasionally produced by it, Dr. Houlton states that in a monastery where the roots had been eaten for supper by mistake, the monks who partook of them were seized in the night with the most extraordinary hallucinations, so that the place became like a lunatic asylum. One monk rang the bell for matins at twelve o'clock at night; of those of the fraternity who attended to the summons, some could not read, some read what was not in the book, and some saw the letters running about the page like so many ants. (*Lancet*, July 6, 1844, p. 479.)

Among the reported cases of poisoning by henbane is the following: A woman collected in a field a quantity of the roots by mistake for parsnips. They were boiled in soup, of which nine persons in the family partook without remarking any particular taste. Very shortly afterwards the whole of these persons felt uneasy, and complained of a bitter acrid taste in the mouth, with nausea. The pupils of the eyes were dilated, and there was indistinctness of vision. These symptoms were followed by great restlessness, convulsions, and continued delirium. The patients successively lost the power of vision, hearing, and voice, and were affected with stupor and insurmountable somnolency. (*Ed. Med. and S. J.*, Oct. 1844, p. 562.) Orfila relates the cases of two men who ate the young shoots of the plant. The first effect was that the earth seemed to pass suddenly from under them; the tongue became paralyzed, and their limbs were cold, torpid, paralyzed, and insensible; the arms were in a state of spasmodic action; the pupils were dilated, the look was fixed and vacant; breathing difficult; the pulse small and intermittent. Besides these symptoms, there was the spasmodic grin (*risus sardonicus*), with delirium; and the jaws were spasmodically closed. Under treatment the men recovered in the course of two days. (*Op. cit.*, 4ème ed., vol. 2, p. 264.)

Mr. White met with the following case: A woman, æt. 34, swallowed, in mistake for a black draught, an ounce and a half of the *tincture* of hyoscyamus, made apparently from the biennial plant, and resembling the pharmacopœial tincture. (The annual plant is said to make a weaker tincture.) In *ten minutes* she had a hot, burning, pricking sensation in the hands, feet, and legs; became giddy and delirious, and complained of great dryness in the throat. Shortly after, in attempting to get out of bed, she found her legs were powerless. A purplish rash appeared over the body, particularly about the neck and face, which were much swollen. The draught was taken at 5 A.M. At 9 A.M. Mr. White found her almost insensible and unable to speak. The tongue was swollen, brown, and dry, and put out with difficulty; the face swollen and scarlet; the pupils were so dilated that the iris was a mere thread-like ring; the skin hot and dry. The poison had been taken on an empty stomach. There was no sickness. In three hours she passed a motion smelling strongly of this drug, but the odor was not perceived in the urine. She could not see distinctly. All motion in the extremities was lost, and their sensibility was diminished. At 4 P.M. she was delirious, and there was sickness; 11 P.M. shivering and coldness of the skin. At 9 A.M. the next day she could see and articulate better. The iris was half a line in breadth. Brandy, opium, ammonia, and other remedies were used, and she gradually improved. It was six days before she acquired a partial use of her legs, and could not then stand without being supported on both sides. She had quite lost her memory, and talked in a rambling manner. She was unable to remember for a minute a single sentence or word she had uttered or read. (*Lancet*, July 5, 1873, p. 8.)

When the extract or decoction is introduced into the rectum, or applied externally to a wound, similar effects are observed to follow. In a case quoted by Orfila, in which a decoction of the plant was used as an injection, the patient suffered from all the symptoms of apoplexy, with the exception of the absence of stertorous breathing.

Appearances.—One fatal case of poisoning by the roots of henbane is quoted by Orfila, and another by the leaves is reported by Wibmer (*op. cit.*, p. 147). The appearances consisted in a general congestion of dark-colored liquid blood in the venous system. The lungs and brain especially manifested this condition. There are commonly no marks of irritation or inflammation in the stomach and bowels.

Fatal Dose.—There are no data by which we can determine the relative activity of henbane. In powder the medicinal dose of the leaves is from five to ten grains; of the seeds, from three to eight grains. The dose of the tincture is from half a drachm to one drachm, and of the extract, from five to ten grains; but this preparation is more likely to vary in strength than any of the others. Dr. Burder states that he has observed great inconvenience to follow from a dose of ten minims of the tincture repeated every six hours. After three or four doses there was pain with oppression of the head. Ten minims given in doses at an interval of six hours, were followed by pain in the head, flashing of light before the eyes, and delirium. (*Lancet*, July 6, 1844, p. 480.) There may be, as in the case of opium, an idiosyncrasy with respect to this drug. Twenty *seeds* have produced complete delirium (Wibmer, *op. cit.*, p. 147), and the same writer states that, in one instance alarming symptoms were caused by seven grains of the extract (p. 154). The poisonous properties of the plant are affected by soil and season. They are most developed in it while the seeds are being formed.

Treatment.—The speedy expulsion of the poison by emetics and castor oil.

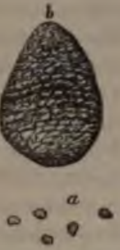
Analysis.—When the vegetable has been eaten, it can be identified only by its botanical characters. The seeds are very small and hard; they are furrowed on the surface, and may be easily confounded with those of belladonna. They are of an oblong, oval, or pyriform shape. In the annexed illustration they are represented magnified (*b*), and of their natural size (*a*) Fig. 60, p. 646. The leaves are peculiar in shape and other characters, by which they may be easily identified. The engraving of the leaf (Fig. 61) is from a photograph of a fresh leaf of henbane.

Hyoscyamia.—The poisonous properties of henbane are known to be owing to a crystalline alkaloidal body, which is called *Hyoscyamia*. It is very difficult of extraction. The crystals have a silky lustre; they are not very soluble in water, but are easily dissolved by alcohol and ether. It has an alkaline reaction, and its saline solutions are precipitated by tannic acid. It has an acrid disagreeable taste resembling that of tobacco. It is highly poisonous, and causes dilatation of the pupils.

Professor Schroff, of Vienna, has performed some experiments on himself and a friend with small doses of this alkaloid. The symptoms produced were giddiness, unsteadiness of gait, with great dryness in the mouth and throat, so that nothing could be swallowed; there was headache, with impairment of the senses of tasting and smelling, and after a time a strong tendency to sleep. The pupils were dilated. With regard to this symptom it was observed that by local application a solution of hyoscyamia produced intense and continued dilatation.

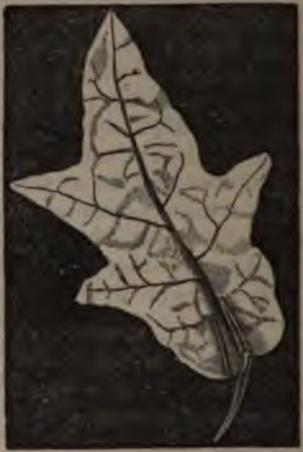
The medicinal dose is assigned by him at from one-sixtieth to one-twentieth of a grain. (*Wochenblatt*, June 16, 1856; *Brit. and For. Med. Rev.*, vol. 19, Jan. to April, 1857, p. 260. See also Reil, *Journal für Toxicologie*, 1857, vol. 2, p. 277.) Dr. Oulmont, *Amer. Jour. Med. Sci.*, April, 1873, p. 528, assigns the medicinal dose at the thirtieth of a grain daily, gradually increased to five or six times that quantity. Its poisonous operation begins to be manifested in a dose of one-tenth of a grain, and the first indications are dryness of the throat and dilated pupils.

FIG. 60.



Seeds of henbane.
a. Natural size.
b. Magnified 30 diameters.

FIG. 61.



Small leaf of henbane, of its natural size and form.

LACTUCARIUM (LACTUCA).

Symptoms and Effects.—The two species of lettuce, known under the names of LACTUCA SATIVA and VIROSA, contain a principle which is possessed of feebly narcotic properties. Orfila has found that the extract, prepared by evaporation at a low temperature, acts upon the brain and nervous system of animals; although very large doses were required for the production of narcotic effects. There is no record of these plants having exerted a poisonous action in the human body.

The inspissated juice of the lettuce is well known under the name of *lactucarium* or *lettuce opium*. (Pereira, *Mat. Med.*, vol. 2, part 2, p. 36.) The Lactuca virosa yields three times as much as the Lactuca sativa; and half a grain of it, according to Dr. Fisher, is equivalent to two or three grains of that obtained from the Lactuca sativa. (*Med. Gaz.*, vol. 25, p. 862.) The juice, when it first escapes, is of a milky-white hue, but, in drying, it forms an extract in small irregular dry masses of a brown color, a bitter taste, and with an odor similar to that of opium. It has a weak narcotic action when given in doses of from five to twenty grains. It varies much in strength. Wibmer found that *two grains* caused headache and somnolency. (*Op. cit.*, p. 200.)

Analysis.—By the smell only it may be mistaken for opium. It is but little soluble in water, and after long boiling it forms a brown turbid solution which produces a greenish tint with a persalt of iron. It therefore contains a little tannic but no meconic acid. On examining a good specimen I have not found any trace of morphia. This shows that an *odor* resembling that of opium may exist in substances which do not contain any meconate of morphia. Nitric acid gives a yellowish tinge to the decoction, as it does to most other vegetable solutions. The aqueous solution is bitter to the taste, which appears to be owing to the presence of a bitter principle called *lactucin,* upon which its feebly narcotic properties probably depend. There are no tests for lactucarium further than the color, the opiate odor, with the want of solubility, and the absence of the other chemical characters of opium. In the plant, it is chiefly combined with malic acid, potash, and resin. (Fisher, *loc. cit.*)

NIGHTSHADE (SOLANUM).

Symptoms and Effects.—There are two species of this plant—the *Solanum dulcamara, Bitter-sweet* or *Woody Nightshade,* which has a purple flower and bears red berries; and the *Solanum nigrum,* or *Garden Nightshade,* with a white flower and black berries. Dunal gave to dogs four ounces of the aqueous extract, and, in another experiment, 180 ripe berries of the Dulcamara, without any ill effects resulting. On the other hand, Floyer states that thirty of the berries killed a dog in three hours. (Wibmer, *op. cit., Solanum.*) These differences may perhaps be reconciled by supposing that the active principle *Solania,* on which the poisonous properties of both species depend, varies in proportion at different seasons of the year. In one instance a decoction of the plant is said to have produced in a man dimness of sight, giddiness, and trembling of the limbs, symptoms which soon disappeared under slight treatment. (For a case of poisoning, by the decoction, see *Med. Gaz.,* vol. 46, p. 548.) Orfila found that the extract of Solanum nigrum had but a feeble effect as a poison; and the fatal cases reported to have been caused by it are perhaps properly referable to belladonna (*Deadly Nightshade*), for which it may have been mistaken. The single death from Dulcamara reported in the Registration returns for 1840, may have been due to a mistake of this kind. In September, 1853, the red berries of the *Woody Nightshade* are stated to have caused the death of a boy, æt. 4, under the following circumstances. He had eaten some of the berries, and at first did not appear to suffer from them; but eleven hours afterwards he was attacked with vomiting, purging, and convulsions, which continued throughout the day; the child being insensible in the intervals. He died convulsed in about twenty-four hours. The vomited matters were of a dark greenish color and of a bilious character. Other children had partaken of the berries at the same time; but one of them suffered only slightly. (*Lancet,* June 28, 1856, p. 715.)

Nevertheless the berries of the Solanum nigrum, on one occasion at least, produced serious effects in three children who had eaten them.

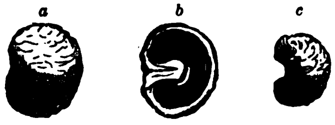
They complained of headache, giddiness, sickness, colic, and tenesmus. There was copious vomiting of a greenish-colored matter, with thirst, dilated pupils, stertorous respiration, convulsions, and a tetanic stiffness of the limbs. One child died in the acute stage; the others died apparently from secondary consequences during treatment. (Orfila, *op. cit.*, 4ème ed., vol. 2, p. 273.) From three to four berries of this plant have been found to produce sleep.

Solania—Analysis.—These plants can be identified only by a botanical examination of the leaves and berries. The active principle in both is an alkaloid, *Solania*, which is itself a poison, although not very energetic. Two grains of the sulphate of solania killed a rabbit in a few hours. The action of solania and its salts upon animals has been investigated by Dr. J. Clarus, of Leipzig. (Reil, *Journal für Toxicologie*, 1857, vol. 2, p. 245.) According to him, solania exalts the sensibility of the skin like strychnia, but destroys life by producing paralysis of the muscles of the chest, like conia or nicotina. It appears to occupy an intermediate place between nicotina and strychnia. It differs from atropia, daturia, and hyoscyamia in not producing stupor or delirium, dilatation of the pupils, or paralysis of the sphincter muscles. He regards it as a narcotico-acrid (cerebrospinal) poison, and assigns the medicinal dose of the acetate for an adult, at one-sixth of a grain. *Tests.*—1. Chromic acid gives a sky-blue color passing to a green. 2. Concentrated sulphuric acid produces an orange-red color passing through a yellow to a violet. 3. Nitric acid with the vapor of ammonia produces a rose-red color. 4. The solution is not precipitated by chloride of platinum or iodide of potassium.

COCCULUS INDICUS (LEVANT NUT).

Symptoms and Effects.—This is the fruit or berry of the ANAMIRTA COCCULUS (*Levant Nut*), imported from the East Indies. The berry contains from one to two per cent. of a poisonous principle (*Picrotoxin*). The shell or husk contains no picrotoxin, but a non-poisonous principle called menispermin. The seeds, in powder or decoction, give rise to nausea, vomiting, and griping pains, followed by stupor and intoxication. There are, so far as I am aware, only two well-authenticated instances of this substance having proved fatal to man. Several men suffered from this poison in 1829, near Liverpool: each had a glass of rum strongly impregnated with Cocculus indicus. One died that evening; the rest recovered. (Traill's *Outlines*, p. 146.) Of the second fatal case, the following details have been published. A boy, æt. 12, was persuaded by his companions to swallow two scruples of the composition, used for poisoning fish. It contained Cocculus indicus. In a few minutes he perceived an unpleasant taste, with burning pain in the gullet and stomach, not relieved by frequent vomiting, as well as pain extending

FIG. 62.



a. Berry of Cocculus indicus, natural size.
b. The same, seen in section with one-half of the semilunar kernel.
c. The kernel, containing picrotoxin.

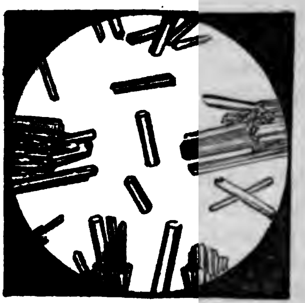
over the whole of the abdomen. In spite of treatment, a violent attack of gastro-enteritis supervened, and there was much febrile excitement, followed by delirium and purging, under which the patient sank on the nineteenth day after taking the poison. On inspection, the vessels of the pia mater were congested with dark-colored liquid blood. There was serous effusion in the ventricles of the brain, and the right lung was congested. In the abdomen, there were all the marks of peritonitis in an advanced stage. The stomach was discolored, and its coats were thinner and softer than natural. (Canstatt, *Jahresbericht*, 1844, vol. 5, p. 298.)

Porter, ale, and beer owe their intoxicating properties in some instances to a decoction or extract of these berries. The fraud is perpetrated by a low class of publicans. They reduce the strength of the beer by water and salt, and then give to it an intoxicating property by means of this poisonous extract. A medical man consulted me some years since, in reference to the similarity of cerebral symptoms suffered by several of his patients in a district in London. It was ascertained that they were supplied with porter by retail from the same house. The effects produced by this drug are remarkable: there is a strong disposition to sleep, and at the same time wakefulness. There is a heavy lethargic stupor, with a consciousness of passing events, but a complete loss of voluntary power. It is a kind of nightmare feeling, altogether different from healthy sleep. Cocculus indicus is sometimes used by robbers to render their victims powerless, and to this form of intoxication the term "hocussing" is applied. It operates readily as a poison on animals, and it has been sometimes used for the malicious destruction of game. In one instance referred to me by the late Sir C. Scott there was reason to believe that 270 young pheasants had been poisoned by grain soaked in a decoction of this substance. *Barber's poisoned wheat* for the destruction of birds owes its poisonous properties to Cocculus indicus. (Horsley.) Poachers occasionally employ it for the purpose of taking fish, which are thereby rendered poisonous. (*Ann. d'Hyg.*, 1843, vol. 1, p. 343.)

The shell appears to have emetic properties. The kernel, which alone contains picrotoxin, is the seat of the poison. The proportion of picrotoxin is about $\frac{1}{100}$th of the weight of the kernel. (See a paper by Dr. Glover, *Lancet*, January 11, 1851, p. 47; and *Ed. Monthly Jour.*, 1851, p. 306. Also another by Dr. J. C. Browne, *Brit. Med. Jour.*, April, 1875, p. 442.)

Picrotoxin—Analysis.—The poisonous principle of the berry of Cocculus indicus crystallizes in slender hexahedral prisms, having a silky lustre. It is soluble in 150 parts of cold water, but is more soluble in boiling water, and the solution has a very bitter taste. When heated in a tube, picrotoxin evolves an acid vapor like digitalin. Hydrochloric acid dissolves it without change of color. It is soluble in alcohol, ether, chloroform, and in amylic alcohol. Sulphuric acid imparts to it an orange-yellow color, which becomes of a pale yellow by dilution. When bichromate of potash is added to the sulphuric acid mixture, green oxide of chromium is produced. Strong nitric acid dissolves it without any change of color. Tannic acid and the chloriodide of potassium and mercury do not precipitate it from its solutions. When boiled with a solution of potash and the sulphate of copper, it reduces the oxide like grape-sugar. It is said, like salicin, to belong to the class of glucosides.

FIG. 63.



Crystals of picrotoxin magnified 124 diameters.

Mr. Langley has shown that this principle may be separated from many of the poisonous alkaloids by taking advantage of its peculiar chemical properties. It does not combine with acids to form salts, but readily with bases. Thus water containing a small quantity of potash will dissolve one-sixth or one-eighth part of its weight of picrotoxin. Water thus alkalized will, it is well known, readily yield most of the alkaloids to ether, when this liquid is shaken with the solution; but if the liquid is strongly acidulated, the alkaloids remain combined with the acid, while the ether shaken with it entirely removes the picrotoxin. Thus, in examining beer supposed to be adulterated with Cocculus indicus, the liquid should be acidulated with hydrochloric acid, and then shaken with two volumes of ether. The ethereal solution thus obtained, when spontaneously evaporated, leaves the picrotoxin in crystals. Mr. Langley states that by this process he has detected so small a quantity as $\frac{1}{750}$th of a grain of picrotoxin in a pint of ale. The stomach of a cat which had been poisoned was treated with alcohol, and the solution evaporated to dryness. Acidulated water was poured on the residue, and the picrotoxin with some organic matter was dissolved. The acid liquid was shaken with ether, and crystals of picrotoxin were obtained by the evaporation of the ethereal solution. (See *Pharm. Jour.*, December, 1862, p. 277.)

One method of detecting this poison in alcoholic liquids, consists in distilling over the alcohol and then testing the extract by physiological or chemical processes. The extract containing Cocculus indicus is intensely bitter, and soon produces on man or on animals stupefying and narcotic symptoms. The extract of a genuine alcoholic liquid loses its narcotic properties when all the alcohol has been separated from it by distillation. For some remarks on this adulteration of beer and other liquids, and a process for separating the poisonous principle, picrotoxin, by amylic alcohol, see *Chem. News,* March 12, 1864, p. 123.

INDIAN HEMP (CANNABIS INDICA).

This substance has been introduced into the British Pharmacopœia in the form of extract and tincture, the dose of the tincture as a narcotic being from five to twenty minims, and of the extract one quarter to one grain. It is estimated that one ounce of the tincture corresponds to twenty-two grains of the extract. The only case of poisoning with Indian hemp with which I have met is the following, which is reported by Mr. Roche. (*Lancet,* 1871, vol. 2, p. 493.)

A lady, æt. 30, suffered from symptoms of poisoning, following a dose of only seven minims of the ordinary tincture. After taking this quantity in a mixture she became drowsy; her vision was dimmed; she was sick; had great thirst, with dryness of the fauces, and slept heavily. Four hours after she had taken the tincture, she was still in a state of narcotism, very drowsy, and not easily roused. The pupils were fully dilated; the eyes suffused; the tongue dry, and the pulse small and quick. These symptoms were followed by profuse perspiration. The next day the symptoms had disappeared with the exception of some dryness of the mouth and feverishness. It will be seen from this case that serious effects were produced by little more than a medicinal dose.

CHAPTER LXVII.

ARTEMISIA ABSINTHIUM—WORMWOOD—OIL OF WORMWOOD—ABSINTHE—BEARDED DARNEL—POISONOUS MUSHROOMS—MISTLETOE—VISCUM ALBUM.

ARTEMISIA ABSINTHIUM—OIL OF WORMWOOD—ABSINTHE.

THE volatile oil extracted from the plant by distillation (*Oleum absinthii*) is of green or greenish-yellow color, and has an acrid bitter taste. It has been long known to have a specific effect over the nervous system, producing headache, giddiness, and other symptoms of cerebral disturbance. Cases of poisoning with it are rare. The following was communicated to me by Mr. W. Smith, of the Chesterfield Hospital.

A druggist's shopman was found early one morning by his master, lying on the floor of the shop, perfectly insensible, convulsed, and foaming at the mouth. As the man had never suffered from fits, and the symptoms were of an alarming character, Mr. Smith was at once sent for. He found him no longer violently convulsed, but insensible; the jaws were clenched, and the pupils dilated. The pulse was weak, slow, and compressible. From time to time he uttered incoherent expressions, and attempted to vomit. With some difficulty Mr. Smith administered to him repeated doses of stimulants, sal volatile and water, lime-water, and an emetic of mustard and sulphate of zinc. Free vomiting ensued, and consciousness partially returned. Artificial warmth was applied to the limbs, and a little brandy and water given at intervals, with draughts of milk and lime-water. He gradually recovered. The matters vomited smelt strongly of oil of wormwood, and the nature of the poison was placed beyond doubt by the discovery of the bottle, with marks on its mouth of the oil having been recently poured out. The druggist stated that at least half an ounce had been taken. From the persistent smell of the oil in the ejected matters, after repeated vomiting, it is probable that this was even less than the real quantity. The man, on recovering, had totally forgotten all the circumstances connected with the case, and persisted in stating that he knew no reason why he should have taken it. It is, however, probable that he imagined himself suffering from worms, and sought relief in an unusual dose of this oil. (See *Ann. d'Hyg.*, 1863, vol. 1, p. 227.)

A French liqueur called ABSINTHE appears to owe some of its properties to the presence of this oil, with a large proportion of alcohol. It has been much used in France, and its effects when taken in excess are those of a narcotic poison. According to Dr. Legrand, it causes derangement of the digestive organs, intense thirst, restlessness, giddiness, tingling in the ears, and illusions of sight and hearing. These symptoms are followed by tremblings in the arms, hands, and legs, numbness in the limbs, loss of muscular power, delirium, loss of intellect, general paralysis, and death.

M. Magnan, who has had under his observation, since April, 1869, two hundred and fifty patients more or less injured in health by the abuse of this intoxicating liquid, and who has besides performed numerous experiments on animals, states that epileptic convulsions are generally observed in these cases. Delirium tremens is the ordinary result of the abuse of alcohol, but the epileptic attacks are specially referable to the absinthe. Magnan describes it as "absinthe epilepsy." (Husemann's *Jahresbericht*, 1872, p. 499, and Bouchardat's *Annuaire de Thérapeutique*, 1872, p. 66.)

Analysis.—One sample of this liquid which I examined had a greenish color, an aromatic odor like that of aniseed, and a hot, pungent, bitter taste. A large quantity of alcohol was obtained by the distillation of it. It acquired a milky appearance on the addition of water, owing to the separation of essential oil. It is a strong alcoholic mixture of oil of aniseed, oil of wormwood, *absinthin*—the bitter principle of wormwood—and some aromatic substances. The oil itself has a bitter taste.

ARTEMISIA SANTONICA—WORMSEED—SANTONIN.

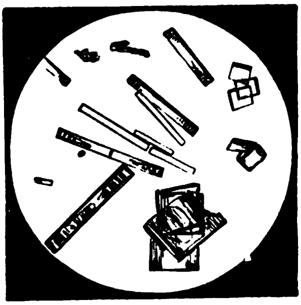
Wormseed is the name by which this substance is generally known. It consists of the unexpanded flower heads of a species of *Artemisia* (*A. santonica*). They resemble seeds. They are used in the treatment of worms in doses of from two to twenty grains. In larger doses this substance produces great irritation of the bowels with cerebral symptoms.

Dr. Linstow, of Ratzeburg, has lately met with the following case. A man gave to his daughter, æt. 10, about 155 grains of wormseed for the cure of worms. In two or three hours she was seized with violent vomiting, followed by convulsions, with coldness of skin. The following day worms were discharged. She was seen by Dr. Linstow on the third day. There was severe vomiting, with convulsions, the pupils were dilated and insensible to light. The girl was drowsy and suffered from some pain in the stomach. She died before any medicines could be employed. The body was not inspected. (*Vierteljahrsschrift*, 1874, vol. 3, p. 81.)

Santonin.—This is a crystallizable neutral principle, which is extracted from wormseed or santonica. The proportion contained in wormseed is from two to two and a half per cent. The dose for an adult is variously given at from one and a half to two grains. In large doses it acts as a narcotico-irritant poison. It affects the bowels as well as the nervous system. The *symptoms* produced are violent vomiting, pain in the abdomen, restlessness, convulsions of the limbs, drowsiness and stupor, the pupils dilated, the pulse frequent and irregular. Some remarkable symptoms are also generally present, namely, colored vision (*chromatopsia*). All objects seen by the eye appear yellow; in some cases they have a violet hue, the complementary color of yellow. (Husemann's *Pflanzenstoffe*, p. 929.)

Analysis.—Santonin has been occasionally mixed with and mistaken for strychnia, or *vice versâ*. It crystallizes in four-sided tables, which have the remarkable property of acquiring a brilliant yellow color by exposure to light (*photosantonin.*) It is not soluble in cold water, and has no taste; but it is soluble in alcohol, and the solution has a bitter taste. Its best solvent is chloroform, four and a half parts of which dissolve one part. It melts at a high temperature, and sublimes in white crystals a few degrees above its melting-point. Nitric, iodic, and sulphuric acids have no action on it. Sulphomolybdic acid produces a pale reddish-brown color. Bichromate of potash added to the mixture with sulphuric acid produces no colors like strychnia, but only green oxide of chromium.

FIG. 64.



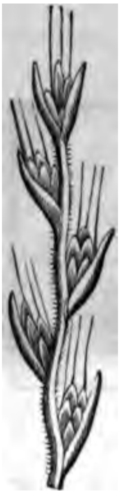
Crystals of santonin, magnified 124 diameters.

BEARDED DARNEL (LOLIUM TEMULENTUM).

Symptoms and Effects.—Poisoning by darnel is generally the result of accident from the admixture of the seeds of this grass with wheat or rye. The seeds are ground into flour and eaten with the bread. From experiments on animals, and from a few observations on man, it appears that the seeds of darnel, whether taken in powder or in decoction, have a local action on the alimentary canal, and a remote action on the brain and nervous system. There is heat, with pain in the stomach, accompanied by nausea, vomiting, and diarrhœa. These symptoms are followed by languor, loss of vision, ringing in the ears, and giddiness. In order to produce such serious effects, the poisonous grain must be taken in a somewhat large dose. So far as I can ascertain, there is no instance recorded of its having proved fatal to man; and as much as three ounces of a paste of the seeds have been given to a dog, without causing death. (Wibmer, *op. cit.*, *Lolium.*)

In January, 1854, Dr. Kingsley, of Roscrea, furnished me with the particulars of some cases in which several families (including about thirty persons) suffered severely from the effects of bread containing, by accidental admixture, the flour of darnel seeds. The persons who partook of this bread staggered about as if intoxicated; there was giddiness, with violent tremblings of the arms and legs, similar to those observed in delirium tremens, but of much greater intensity (the patients requesting those about them to hold them, and experiencing great comfort from this assistance being given to them); greatly impaired vision, every object appearing of a green color to the sufferer; coldness of the skin, particularly of the hands and feet; great prostration of strength, and in several cases vomiting. Under the free use of stimulants and castor oil the whole of the patients were convalescent on the following day, but much debilitated from the effects of the poison.

FIG. 65.



Lolium temulentum, or bearded darnel.

In one instance in which darnel seeds were mixed in the proportion of one-tenth part with rye, the persons partaking of the bread suffered from giddiness, headache, nausea, vomiting, deafness, and cramps. (*Medical Gazette*, vol. 45, p. 872; *Ann. d'Hyg.*, 1853, vol. 2, p. 147.) Among the symptoms in other cases there has been noticed a sense of burning in the mouth and throat, with confusion in the head, trembling, and a small irregular pulse. (See *Ed. Monthly Jour.*, Aug. 1850, p. 180.) When these symptoms attack simultaneously many persons who partake of the same bread, there is strong ground for suspicion.

An accident occurred in Germany from the seeds of the darnel becoming mixed with those of barley which was made into bread. The prominent symptom was giddiness in a severe form. As a result of this mixture the poisonous principle of darnel (which is capable of resisting a baking heat) may find its way into beer or brandy. (Casper's *Vierteljahrsschrift*, Oct. 1857, p. 343.) A wet season is favorable to the growth of darnel with the different varieties of corn. The seeds are difficult of separation.

Analysis.—This plant may be recognized by its botanical characters. Pfaff examined darnel in order to discover a poisonous alkaloid, but there was no trace of such a substance. By distillation with water he obtained two kinds of ethereal oil, one lighter and the other heavier than water; they were colorless, but had the odor of fusel oil.

POISONOUS MUSHROOMS (FUNGI).

Cases of poisoning by mushrooms (FUNGI) are by no means unusual as the result of accident. According to Dr. Badham there are five thousand recognized species, of which only a few can be safely eaten. Among them the *Agaricus compestris* and *esculentus* are perhaps most commonly employed as articles of food. It is a curious fact that the poisonous properties of mushrooms vary with climate, and probably with the season of the year at which they are gathered, as well as the locality. Another circumstance deserving of notice is, that by idiosyncrasy, some persons are liable to be seriously affected even by those species which are usually regarded as innocent. Some species which are poisonous in this country are used freely by the Russians; it appears they are in the habit of salting, boiling, and compressing them before they are eaten; and this may in some instances suffice to account for their having no noxious effects. Dr. Badham states that the *Agaricus campestris*, or common mushroom, which is largely eaten in England, is regarded as poisonous in Rome, and is accordingly rejected; while some varieties, which in this country would produce symptoms of poisoning, are eaten in Italy with impunity. There do not appear to be any satisfactory rules for distinguishing the mushrooms which are wholesome from those which are poisonous. The best test is that assigned by Dr. Christison, namely, that the poisonous vegetable has an astringent styptic taste, as well as a disagreeable, but certainly a pungent odor. All mushrooms that are highly colored, or grow in dark and shady places, are generally poisonous.

Symptoms and Appearance.—The noxious species of mushrooms act sometimes as narcotics, at others as irritants. It would appear from the reports of several cases that when the narcotic symptoms are excited, they come on soon after the meal at which the mushrooms have been eaten, and that they are chiefly manifested by giddiness, dimness of sight, and debility. The person appears as if intoxicated, and there are singular illusions of sense. Spasms and convulsions have been occasionally witnessed among the symptoms when the case has proved fatal. Dr. Peddie has related three cases of poisoning by mushrooms (*Edin. M. and S. J.*, vol. 49, p. 200), in which the poison acted as a pure narcotic; there was no pain in the abdomen, nor irritation in the alimentary canal. The narcotic symptoms began in half an hour with giddiness and stupor; the first effect with one patient was, that every object appeared to him to be of a blue color. The three patients recovered, two of them rapidly. When the drowsiness passes off, there is generally nausea followed by vomiting; but sometimes vomiting and purging precede the stupor. If the symptoms do not occur until many hours after the meal, they partake more of the characters of irritation, indicated by pain and swelling of the abdomen, vomiting, and purging. In a recent case of poisoning by mushrooms, there was slight vomiting about an hour and a half after the meal, but no violent symptoms until after the lapse of ten hours. Several cases, in which the symptoms did not appear until after the lapse of fourteen hours, are reported in the *Medical Gazette* (vol. 25, p. 110). In some instances the symptoms of poisoning have not commenced until thirty hours after the meal, and in these, narcotism followed the symptoms of irritation. It might be supposed that these variable effects were due to different properties in the mushrooms; but the same fungi have acted on members of the same family, in one case like irritants, and in another like narcotics. In most cases recovery takes place, especially if vomiting is early induced. In the few instances which have proved fatal, there has been greater or less inflammation in the stomach and bowels, with congestion of the vessels of the brain. (See *Med. Gaz.*, vol. 46, p. 307; vol. 47, p. 673; and *Jour. de Chimie Med.*, 1853, p. 694.) Balardini states that, of sixty-eight cases of poisoning by mushrooms which occurred in the province of Brescia, during a period of twenty years, twenty proved fatal. The principal symptoms were nausea, uneasiness, and pain in the abdomen, giddiness; a state resembling intoxication; vomiting and purging; loss of the power of locomotion, with convulsions. (Canstatt's *Jahresbericht*, 1844, v. 300.) In some persons even the edible mushrooms will produce disorder of the stomach and bowels as a result of idiosyncrasy. In six cases which occurred to Dr. Keber, in which the *Helvella esculenta* had caused symptoms of poisoning, the principal symptom was urgent vomiting, and the patients were jaundiced as soon as the vomiting had ceased. One patient, a girl of 18, fell into a state of coma, from which she did not recover for three days. It was probable that, in this instance, the noxious effects were due to season. (*Gaz. des Hôp.*, Oct. 10, 1846.) The common truffle (*Morchella esculenta*) has been known to cause severe symptoms of irritant poisoning. In some of these cases it is probable that the truffles had undergone decomposition before they were eaten. (*Ed. Med. and Surg. Jour.*, Oct. 1845, p. 530, and *Ann. d'Hyg.*, 1845, vol. 1, p. 214.)

In the *Lancet* of June 28, 1856, p. 716, a case is related in which some poisonous fungi accelerated death, if they did not actually cause it. The man had eaten stewed mushrooms and died rather suddenly, having shortly before complained of pain in the bowels. The mucous membrane of the gullet as well as that of the stomach was inflamed.

Dr. Porter Smith (Sept. 1872) communicated to me the following case: An aged woman ate some edible mushrooms (as she believed) about 9.30 A.M. They were quite fresh, and eaten in a cooked state. In about an hour she suffered from pain in the abdomen and violent vomiting, followed by stupor. Dr. Smith saw her at 1 P.M. She was drowsy, and unable to speak. There was paralysis of the left arm and leg, with a puffing up of the cheeks with each breath. The pulse was 60. In spite of treatment she sank and died at 7 A.M. the following morning, about twenty-two hours after eating the mushrooms. There was no inspection of the body, and no clear evidence of the nature of the mushrooms eaten.

Poisoning with mushrooms is usually the result of accident or mistake. They are not taken for the purpose of suicide, and I have met with only one instance in which it was alleged they were intentionally given to destroy life. In 1873, a gardener in the metropolitan district was tried on a charge of manslaughter for causing the death of a young woman by giving her poisonous mushrooms. The accused, it was alleged, had a motive for the act, but he denied that he knew the mushrooms to be poisonous. The deceased fried them, and had some for breakfast. . She suffered severe pain, and died the same evening. Other persons who partook of them were also taken ill, but recovered. There was an absence of proof to show that he knew them to be noxious, and he was acquitted. This form of homicide would be very difficult to establish. It would be necessary to show that the mushrooms were really poisonous, and to the knowledge of the accused. None might be forthcoming, so that there would be no botanical evidence of their poisonous nature. But as persons have died from taking edible mushrooms, it might be alleged that there was nothing criminal in the act, and that death was owing to idiosyncrasy.

In the *Guy's Hospital Reports* for October, 1865, p. 382, I have recorded two fatal cases, in a mother and daughter, who died from the effects of the *Amanita citrina*, a yellow-colored fungus, gathered in mistake for mushrooms. The woman fried the fungi, and they were eaten for supper. No symptoms appeared for seven hours. The child when seen by a medical man was feverish and thirsty, and the pupils were strongly dilated. There was severe pain in the stomach, and a sense of constriction in the throat. The child became convulsed and insensible, and died forty-one hours after eating the fungi. The mother and another child suffered from similar symptoms; the mother partially recovered, but had a relapse, and died on the fifth day. No inspection of the bodies was made.

Mr. Sadler was about to deliver a lecture on edible and poisonous fungi in Edinburgh, when he accidentally swallowed a quantity of the spores of a large species of puff-ball (*Lycoperdon giganteum*). In an hour and a half he was very ill, and suffered from violent pain in the abdomen. This pain did not subside until after nine days. The gastric irritation was attributed by Sir R. Christison to the spores of the fungus. The puff-ball is edible in its young state, but its matured spores should be avoided. (*Brit. Med. Jour.*, 1874, vol. 1, p. 595.) Dr. Drummond met with a case in which a lady ate two or three fine genuine mushrooms in a cooked state. In an hour and a half she had spasmodic pain in the bowels, with severe and well-marked tonic spasms of the muscles of the arms, head, and neck. She was relieved by an emetic and brandy and water. She recovered, but for some weeks had numbness and stiffness of the muscles of the arms, with difficulty of articulation. (*Brit. Med. Jour.*, Oct. 24, 1874, p. 524.)

Mr. Taylor, of Emsworth, relates a fatal case of poisoning by fungi which was attended with symptoms of irritation resembling those caused by arsenic. There was no loss of consciousness or sensibility. G. F., æt. 13, fried and ate for breakfast at 8.30 A.M., two fungi which he had found growing under a tree. He returned to his work without complaint. At 12 he had his dinner of pork and vegetables. At 1 P.M. he returned to work, where he remained until 6 P.M., working the whole time without any complaint. Soon after he reached home, he complained of feeling ill and vomited violently. Purging then followed, with severe spasmodic pain in the abdomen. These symptoms continued throughout the night until 6 A.M. The bowels then ceased to act. Mr. Taylor saw him at 11.30 A.M. He was then suffering from constant pain in the bowels, occasionally aggravated; there was tenderness over the abdomen generally, but especially over the course of the transverse colon, with vomiting every ten minutes; great thirst, skin warm and perspiring, pulse ninety, and great depression. At 3 A.M. on the second day he was again seen. Vomiting and purging had returned. There was great exhaustion; pulse imperceptible; the action of the heart feeble. He was lying in bed on his back, with the knees drawn up. Sensibility and consciousness were perfect. He complained of great pain in the stomach; there was tenderness over the abdomen, but no swelling of the cavity. In another hour he died, *i. e.*, about forty-four hours after eating the fungi, and about thirty-four after the first setting-in of the symptoms. Others partook of the fungi, but in small quantity, and they did not suffer. On inspection, the heart on the right side contained a little fluid blood. The left ventricle was contracted and empty. The lungs were healthy, and there was only cadaveric congestion. The lining membrane of the stomach and small intestines was throughout injected, the bluish-red appearance diminishing in intensity as it approached the cæcum. There were a few ecchymosed patches near the intestinal end of the stomach. The organ contained six ounces of a brownish liquid resembling thin gruel. The large intestines were empty and pale, and the spleen was congested; the other organs were healthy. (*Med. Times and Gaz.*, Nov. 21, 1863, p. 536.) In many of its features, and in the absence of narcotic symptoms, this case resembled one of acute poisoning by arsenic. The fact that nearly ten hours elapsed before the symptoms of irritation commenced, and that there was no blood in the matters discharged by vomiting and purging, were the most marked differences.

In August, 1871, two children died at Flushing, near Falmouth, from the effects produced by noxious fungi. Several other persons were placed in a precarious condition from the same cause. Some fowls died from eating portions of the mushrooms. Among the cases of poisoning by fungi reported in Husemann's *Jahresbericht* (1872, p. 534), are the following: A man, æt. 43, and his daughter, æt. 5, suffered severely from eating the *Amanita pantherina*. The earliest symptoms appeared in two hours and a half after the meal. They were thirst, faintness, delirium, nausea, paleness of the face, and cold extremities. After eleven hours, there was stupor, with tenderness of the abdomen. In the child, there was cyanosis of the legs with contracted pupils. It was remarked that, even fourteen hours after the fungi had been eaten, portions of them were discharged by vomiting from the action of emetics. They both recovered.

In a case reported by Dr. Stevenson, the *Agaricus stercorarius* was the cause of the symptoms. These were chiefly referable to the nervous system: oppressed breathing, severe pain across the forehead, dimness of sight, and giddiness. The man staggered in walking, and had some difficulty in keeping himself upright. In three hours he was in a state of profound stupor, with pupils dilated and inactive, and the pulse slow and feeble. There were convulsive twitchings of the nervous system. He did not suffer any pain in the stomach or bowels. He soon recovered. (*Guy's Hosp. Rep.*, 1874, p. 419.) It is strange that, with such facts as these occasionally presenting themselves, educated persons can be found who persist in denying that mushrooms are under any circumstances poisonous. In a period of five years (1865–7) six deaths were recorded to have taken place from eating poisonous fungi.

Ketchup, a liquor made from mushrooms, has occasioned faintness, nausea, and severe pain in the abdomen, disappearing only after some hours. (*Dub. Med. Press*, Sept. 24, 1845, p. 195.) There are two modes of explaining this effect: 1, either that the person labors under an idiosyncrasy with respect to mushrooms in general; or, 2, that noxious have been gathered by mistake for esculent mushrooms. A case is on record which shows that a medical jurist may be easily misled when any active poison is mixed with and administered in a dish of mushrooms. A servant girl poisoned her mistress by mixing arsenic with mushrooms. This person died in twenty hours, after suffering severely from vomiting and colicky pains. On dissection, the stomach and intestines were found inflamed. Death was ascribed to the effects of the mushrooms, which were considered to have been unwholesome; and the fact of poisoning with arsenic only came out many years afterwards, by the confession of the prisoner. This shows with what a watchful eye such cases should be examined; in the absence of an analysis of the contents of the stomach, it would be impossible to develop the truth.

Treatment.—The free use of emetics and castor oil.

Analysis.—The discovery of portions of the mushrooms undigested in the matter vomited, or a description of the food eaten, will commonly lead to a recognition of this form of poisoning. The poisonous principle contained in mushrooms has been called *Fungin*, although this name has been also given to the soft spongy substance of which the mushroom consists. Fungin is described as a volatile substance, soluble in cold water, and readily extracted by hot water. Hence some varieties of noxious mushrooms may be eaten with impunity when they have been well boiled in water, and afterwards pressed. One of the most poisonous in this country, *Agaricus muscarius*, or Fly-mushroom, renders the water in which it is boiled so poisonous that animals are killed by it, while the boiled fungus itself has no effect upon them. The liquid procured from it is used as a fly-poison, whence the name of the mushroom is derived. It is an autumnal fungus, known by its rich orange-red color. But as it is well known that, in spite of cooking at a high temperature, many of these fungi have destroyed life, the theory is not altogether consistent with facts.

MM. Sicard and Schoras affirm that the poisonous principle in many species of mushrooms is an alkaloid, as it unites with acids to form salts, which are extremely poisonous. The poison was rapidly fatal to frogs, and a small quantity was sufficient to kill a dog. Its effects on animals, according to them, were similar to those produced by *curarina*. (*Dub. Med. Press*, Nov. 1865.)

Dr. Brunton has described a principle which he detected in the *Agaricus muscarius*, called *Muscarin*. It appears to act as a cardiac poison, stopping the action of the heart, especially in small animals, such as the frog. He also states that atropia has an antagonistic effect to muscarin. This principle has been found to produce an action on the lungs, amounting to intense difficulty of breathing, by causing spasmodic contraction of the pulmonary vessels. (*Brit. Med. Jour.*, Nov. 14, 1874, p. 617.)

The fungi can be recognized only by their special botanical characters. An experienced mycologist (the Rev. J. Berkeley) says, "No general rule can be given for the determination of the question whether fungi are or are not poisonous. Color is quite indecisive, and some of the most dangerous fungi,—and amongst them the *Agaricus phal-*

loides,—are void of any unpleasant smell when fresh, although the most wholesome may be extremely offensive when old. Experience is the only safe test, and no one should try species incautiously with whose character he is not thoroughly acquainted." The learned mycologist who gives this advice appears to have forgotten that a person may lose his life in making this "thorough acquaintance" with the characters of fungi. Portions of the fungi may be found in the food or in the contents of the stomach, but if there has been vomiting and purging, it is probable that the whole of the substance may have been expelled before death. Fungi contain but little solid matter.

Some poisonous fungi which were exposed for sale in open market have been properly seized and condemned as unfit for human food. Much has been said and written on the methods of distinguishing the edible from the noxious fungi, but instances have occurred in which the former have produced symptoms of poisoning and have destroyed life. A case in which a woman died in twenty hours from eating ordinary mushrooms was communicated to me by Dr. H. P. Smith, of Shepton Mallet, in August, 1873. The symptoms resembled those above described.

THE MISTLETOE (VISCUM ALBUM).

Mr. Dixon, of Whitehaven, met with the following case: In December, 1873, he was called to see a boy, æt. 14, who had been seized suddenly with alarming symptoms. About an hour previously the boy had left the house apparently quite well, but in forty minutes he was found lying in the street in a state of insensibility. Mr. Dixon saw him in about a quarter of an hour. The first impression produced on his mind was that the boy was intoxicated. His countenance was suffused, the lips were livid, the conjunctivæ injected, the pupils slightly dilated and fixed; the breathing slow and stertorous. On pricking the soles of the feet, the limbs were quickly drawn up, showing that there was no paralysis of the excitomotory functions. The odor of the breath gave no evidence of alcohol. Cold affusion was employed. He was soon able to speak, but talked incoherently, and was inclined to be violent. He had spectral illusions. Emetics were given, and these brought away eight partially masticated berries of the mistletoe. He continued in a state of excitement for two hours, after which he fell asleep. On the following morning he had quite recovered. He said he ate the berries of the mistletoe at about 8.30 P.M., soon after which he began to feel giddy, and from that time he had no recollection of what had transpired. He had taken no spirit or alcoholic liquid. (*Brit. Med. Jour.*, Feb. 21, 1874, p. 225.)

Wibmer states that the berries contain no poisonous matter, and it appears that birds eat them with impunity. Nevertheless, judging from this case, they have a decided action on the brain.

SPINAL POISONS.

CHAPTER LXVIII.

POISONING BY NUX VOMICA—SYMPTOMS—APPEARANCES—FATAL DOSE AND PERIOD OF DEATH—ANALYSIS—NUX VOMICA BARK—ST. IGNATIUS'S BEANS—UPAS TIEUTE—BRUCIA—AKAZGA—VERMIN-KILLERS.

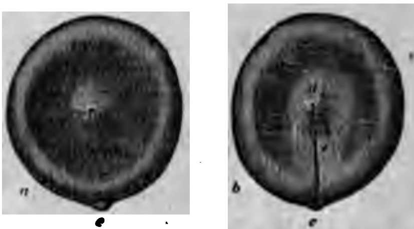
Remarks.—The poisons belonging to this section are so named from the fact that their chief action is exerted on the spinal marrow, the brain being unaffected or only secondarily affected. With an exaltation of sensibility there are the most violent convulsions, in which the muscles become rigid and fixed, producing a state of the body resembling tetanus. There is no stupor or delirium; consciousness is generally retained until just before death. *Strychnia* is the poisonous alkaloid which produces these remarkable effects. It was discovered in 1818, in the seed of nux vomica, by Pelletier and Caventou. It is peculiar to plants or seeds grown in tropical climates, and has hitherto been found in five only.

NUX VOMICA.

This is the seed of the *Strychnos Nux Vomica*, which is a native of Coromandel, Ceylon, and the jungles of Bengal. The seed has the shape of a flat, round kernel, of a grayish-brown color, of about the size of a shilling, but much thicker. It is covered with a fine silky fibrous down, radiating from the centre, which is raised on one side and depressed on the other (see Figs. 66, 67, p. 662). The fruit of the tree which yields it is said to be of the size of a pear, and to contain from three to five of these seeds in the midst of a pulp. As in the case of the cherry-laurel (p. 592), the pulp is described as not being poisonous, while the seeds contain *strychnia*, one of the most deadly poisons known. One seed weighs about thirty grains in the dry state. The proportion of strychnia contained in the seeds has not been accurately determined. Gmelin has assigned the proportion at (0.4) less than one-half per cent. (*Chimie Organique*, p. 111), but Mr. Horsley considers it to amount to one per cent. by weight. The strychnia is combined with a vegetable acid, the strychnic or igasuric acid, and this renders the alkaloid soluble in water. There is another poisonous alkaloid associated with strychnia in the seed, namely, *brucia*. This acts upon the body like strychnia, but with about only one-sixth of the power. In addition to these poisonous alkaloids, the seed contains woody fibre, gum, wax, and oil.

Nux vomica is commonly sold to the public in the form of a grayish-brown powder, at the rate of eight pence an ounce. In this state it may be mistaken for the powders of numerous medicines—ipecacuanha, cinchona bark, etc.; but it is known by its intensely bitter taste, which is persistent, and by the fact that, owing to the presence of brucia, it strikes a deep orange-red color when treated with strong nitric acid. Most medicinal powders give, with this acid, a dingy green or brown color. Nux vomica powder may, however, in the process of grinding, be mixed with other innocent powders, which will entirely destroy or mask this chemical reaction. On one occasion I found the nux vomica to be strongly impregnated with the powder of guaiacum, so that nitric acid produced with it a deep green color. This fact served to identify the sample, and it was proved that it had derived this impregnation from its having been ground in a mill in which guaiacum had been previously ground. In other cases the powder may be mixed with farinaceous substances. Nux vomica, if used as a poison at all, is employed in the form of powder. Its chief use is for the destruction of vermin. It has, however, occasioned numerous deaths, chiefly as a result of suicide. Owing to its bitter taste, its presence in an article of food would be detected in any attempt at murder. In the case of Wren (p. 665) the poison was mixed with milk by the prisoner, an ignorant lad; but it completely altered the color and taste of the milk, and thus led to detection.

FIG. 66. FIG. 67.



Seeds of nux vomica, natural size. *a*, convex surface; *b*, concave surface; *c*, hilum or umbilicus.

Symptoms.—At a period varying from a quarter of an hour to an hour, or even longer, after the poison has been swallowed, the patient experiences a sense of uneasiness or restlessness, and suddenly loses the power of walking, standing, or moving; he is then seized with twitchings of the muscles, followed by shocks or tetanic spasms, affecting the whole of the muscular system; the body becoming rigid, the limbs stretched out, and the jaws so fixed that considerable difficulty is experienced in introducing anything into the mouth. The muscles of the face are fixed by spasmodic contraction producing the sardonic grin, and the body sometimes assumes the state of opisthotonos; there is consciousness, and the intellect is generally clear. This spasmodic state ceases for a time, but after a short interval reappears; and the chest may become so fixed by spasms of the intercostal muscles as to give the idea of impending suffocation. After repeated attacks, generally increasing in severity, the patient dies either suffocated or exhausted. Drowsiness, giddiness, and a feeling of general illness have in some instances preceded the spasms; vomiting, pain in the abdomen, and other symptoms of irritation, have also been occasionally witnessed among the symptoms.

A woman, æt. 23, swallowed 120 grains of powdered nux vomica, mixed with water. Some time after, she was suddenly seized with giddiness, a loss of power in her legs, and a general feeling of stiffness in the body, especially in the neck. She fell, was carried home, and was then seen by a medical man. He found her with her face flushed, pupils dilated, the pulse quick as well as the breathing; numbness and stiffness of the legs, with a feeling of constriction across the chest. Every two or three minutes there was a convulsive tetanic spasm throughout the body, but it was only momentary, like an electric shock. The stomach-pump was used and other remedies were applied, under which she recovered, suffering only from a slight stiffness of the jaws and debility. (*Lancet*, Dec. 15, 1849.) In another case, in which a similar dose was taken by a man, the first symptoms were profuse perspiration, with twitchings of the muscles, ending in a strong and general tetanic spasm. The mind was clear, and questions were answered rationally during the intervals of the spasms. It was observed that these were brought on when any attempt was made to wipe the perspiration from his face. (*Lancet*, July 5, 1856, p. 11.) Two hours after he had taken the poison, an emetic of sulphate of zinc was given to him, and it produced violent vomiting. The tetanic spasms then gradually subsided. A man, æt. 20, swallowed 90 grains of the powder. Spasms of the muscles appeared in ten minutes. In three-quarters of an hour he was in a profuse perspiration, the skin of the head and face congested, the eyes suffused, the pupils slightly contracted, and the pulse hard. Fits of tetanus, each lasting about half a minute, then attacked him. All the muscles were rigid, and his breathing appeared for the time suspended. The muscles were then relaxed, and he was able to answer questions. In two days he recovered. (*Lancet*, Oct. 22, 1853, and *Med.-Chir. Rev.*, Jan. 1854, p. 292.) The spasm does not always involve the chest. A youth, æt. 19, swallowed a teaspoonful (= 65 grains) of the powder of nux vomica in a cup of cocoa; in fifteen minutes he was attacked with convulsions, and fell from his seat. The whole of the body was affected with convulsive twitchings, but these were unattended with pain. The chest was not affected, and there was no difficulty of breathing. He felt the approach of the spasms, but could not describe the sensation. The stomach-pump was used with benefit, and the symptoms disappeared in three hours. (*Med. Times and Gaz.*, April 28, 1855, p. 424.)

A physician took by mistake five grains of the alcoholic extract of nux vomica in two pills, and his wife took a similar dose at the same time. They had tea, and felt no ill effects for forty minutes, when the physician in rising to go to the door, suddenly exclaimed, "Hold me!" The wife rose to render assistance, but she was suddenly fixed in her position by muscular spasm. In ten minutes they were seen by a medical man, who prescribed emetics, which acted speedily and powerfully. The two patients were fixed by spasm to the chairs on which they were sitting, the convulsions coming on at intervals, and being rapidly succeeded by a relaxation of the muscles. During the fit, the heads were drawn backwards, there was spasmodic clenching of the teeth, the heels were fixed to the ground, the eyes protruded from their sockets, and both patients exclaimed, "Hold me! hold me!" although there was a person on either side of each. In about five hours, under the use of emetics, the spasms subsided. On the next day they recovered, but suffered from some debility. (*Med. Times and Gaz.*, Jan. 16, 1858, p. 69.) A boy, æt. 12, put into his mouth about eight grains of the extract. Finding it bitter he spat it out. He was admitted into Guy's Hospital, under Dr. Fagge, suffering from tetanic spasms, difficulty of breathing, and other symptoms of poisoning. His face was flushed, the pupils were dilated, the pulse rapid, the jaws were not affected, and the boy was quite conscious. Spasms were brought on by merely touching him; there were distinct intermissions. The symptoms subsided under treatment, and he left the hospital on the third day. No poison was found in the vomited matters; but from five ounces of urine passed, five hours after the poison had been taken, Dr. Stevenson obtained two stains, one of which gave the color reaction for strychnia, and the other for brucia. (*Guy's Hosp. Rep.*, 1869, p. 265.) The subject of poisoning by nux vomica, including a collection of cases, has been ably treated by Dr. Husemann, in Reil's *Journal für Toxikologie*, 1857, vol. 2 H., p. 469.

Chronic Poisoning.—Medicinal doses frequently repeated may produce all the effects of chronic poisoning. A lady took three grains of the powder of nux vomica thrice daily, for sixteen days (= 144 grains in the whole). There were no obvious effects for a fortnight, when there was purging with colicky pains. The nux vomica was withdrawn, and on the fifth day after its withdrawal, the patient suffered from ringing in the ears, drowsiness, increased sensibility to light and sound, numbness, and impairment of speech. On the ninth day she lost her speech; tetanic symptoms, with twitchings of the muscles of the face and arms, set in, as well as fixation of the jaws (trismus). There were slight intervals of relaxation, during which she swallowed with difficulty. The pupils were dilated and the skin was hot. The spasms increased, and on the twelfth day the breathing became affected. In the evening of this day, she was seized with a strong tetanic convulsion, in which respiration ceased, the face became livid, the brows contracted, the lips were drawn widely apart, and the features greatly distorted, assuming the sardonic grin. During the night she had four similar paroxysms, and died apparently exhausted on the twenty-eighth day after she had commenced with the nux vomica, and on the twelfth day after its discontinuance. (*Lancet*, June 14, 1856, p. 654.)

Appearances.—These are not very characteristic. The body is usually found rigid. There is congestion of the brain and its membranes, with engorgement of the lungs. The heart has been found, in some instances, empty and flaccid (see *Med. Times and Gaz.*, 1856, Feb. 9, p. 149); while in others it was distended with dark-colored and fluid blood. The mucous membrane of the stomach and intestines is occasionally congested. The powder has been found adhering tenaciously to this membrane. The spasmodic state of the muscles has continued for some time after death. In the fatal case of chronic poisoning above referred to, there was congestion of the membranes of the brain, and the heart was contracted and empty. There was a slight inflammatory appearance in the ileum. (*Lancet*, June 14, 1856, p. 654.)

Fatal Dose—Period of Death.—The medicinal dose of nux vomica powder is from two to three grains, gradually increased. A long-continued use of it may produce, apparently as a result of accumulation, the effects of chronic poisoning (*supra*). Large doses are said to have been given, on some occasions, with impunity. Nux vomica is used in pharmacy in the form of extract and tincture. The dose of the former is from half a grain to two grains, and of the latter from ten to twenty minims.

The smallest fatal dose yet recorded is *three grains* of the alcoholic extract of nux vomica. Two cases of poisoning occurred in London, in 1839, in each of which fifty grains of the powder proved fatal. In one of these, death took place in *an hour;* the druggist who sold the poison said that he did not think a dose of fifty grains was sufficient to cause death! but a smaller quantity has been known to destroy life. In an old case reported by Hofmann (1739), and quoted by Christison (p. 901), also by Traill (*Outlines*, p. 137), *thirty grains* of the powder, in two doses of fifteen grains each, proved fatal. The poison was given by mistake to a girl, æt. 10, laboring under quartan fever, and *summis anxietatibus præcedentibus, et ad vomendum conatibus*, she soon afterwards died. This is, I believe, the *smallest* fatal dose of the powder recorded. It is about equivalent to the weight of one full-sized seed, and to only one-third of a grain of strychnia in the two doses. The quantity of nux vomica required to destroy life became of some importance in *Reg.* v. *Wren* (Winchester Spring Assizes, 1851). The prisoner was convicted of an attempt to administer this poison in milk; the quantity separated from the milk amounted to forty-seven grains, which was above a fatal dose. The intense bitterness which the nux vomica gave to the milk led to detection, and this would, in general, be a bar to the criminal administration of this poison, except in the form of extract in pills. In a case which occurred to M. Pellarin, a man swallowed about 300 grains of nux vomica, and no symptoms appeared for two hours. He then died rapidly in a violent convulsive fit. (*Ann. d'Hyg.*, 1861, vol. 2, p. 431.)

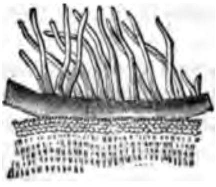
Death may occur in from one to twelve hours; but Dr. Christison quotes a case in which a man died in *fifteen minutes* after taking a dose (*Op. cit.*, p. 898). This is probably the shortest period. There are many instances of recovery on record in which early treatment was resorted to. Mr. Iliff has reported a case in which a woman recovered after taking two drachms of the powder. (*Lancet*, Dec. 15, 1849.) Sobernheim mentions the case of a young man, who took half an ounce of the powder, and suffered from the usual symptoms; emetics were administered and he recovered. A second occurred to Dr. Basedow, of Merseburg. A young lady swallowed, by mistake, a tablespoonful

(=360 grains, or three-quarters of an ounce) of the powder; she soon lost the power of walking, and fell down, but did not lose her recollection. There was great difficulty of breathing. Emetics were administered with good effect, and she recovered. A third case is described by Mr. Baynham, of Birmingham. A girl, æt. 20, swallowed half an ounce of the powder. In half an hour the usual tetanic symptoms came on. She was perfectly sensible. In administering remedies, the spasm of the muscles of the jaw was such as to cause her to bite through the cup. The convulsions gradually subsided in about four hours from the first attack; and on the next day, although feeble and exhausted, she was able to walk home. (*Med. Gaz.*, vol. 3, p. 445.) The reporter of this case states that he has often prescribed a scruple of powdered nux vomica daily, without any injurious effects following! For another case of recovery, in which half an ounce was taken, see *Prov. Med. Journal*, Jan. 7, 1846, p. 5; and for a case in which this dose proved fatal in seven hours, see *Lancet*, May 17, 1856, p. 551. Husemann has collected forty-one cases of poisoning by nux vomica. (Reil's *Journal*, 1857, vol. 4 II., p. 521.)

Treatment.—The removal of the poison from the stomach by emetics, or the use of the stomach-pump, must be chiefly relied on. Unless these means are employed early, the jaw may become spasmodically fixed, so as to render all efforts at relief unavailing. In general, however, the spasms have intermissions, so that there may be time to apply remedies in the interval. The free use of emetics has been attended with great benefit. It has been asserted that vomiting does not occur in this form of poisoning, but this is an error.

Analysis.—The seed of nux vomica is hard, brittle, tough, and difficult to pulverize. The powder is of a grayish brown color, like that of liquorice; it is sometimes met with in a coarsely rasped state; it has an intensely bitter taste. It yields to water and alcohol—strychnia, brucia, igasuric or strychnic acid, and some common vegetable principles. Heated on platinum-foil, it burns with a smoky flame. It is colored brown by a solution of iodine. Nitric acid turns it of a dark orange-red color, which is destroyed by chloride of tin. The aqueous *infusion* or decoction is reddened by nitric acid, and is freely precipitated by tincture of galls. Persulphate of iron gives with it an olive-green tint. These properties are sufficient to distinguish it from various medicinal powders which it resembles.

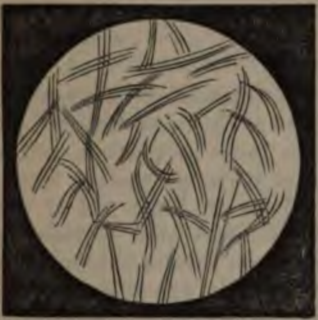
FIG. 68.



Magnified view of a section of nux vomica, showing the hairs projecting from the surface (Pereira).

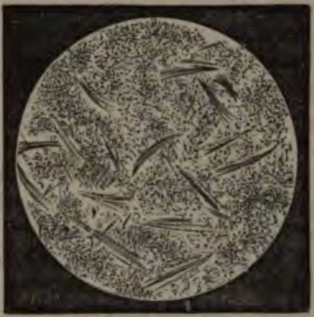
The fine silky fibres which cover the surface of the seed may be sometimes detected in the powder and identified by the microscope. For this purpose a small quantity, moistened with water, should be examined with a power of about 70 to 100 diameters. We may then be able to perceive fragments of the silky fibres intermixed with opaque and irregular portions of the powder. Their appearance with and without the powder is indicated in the two figures (69, 70) annexed. The proportion which the fibres bear to the whole nut is so small that we may be obliged to examine two or three samples before we find any. A solution of iodine gives to the fibres a golden-yellow color. There are no starch-granules to be seen in the genuine powder. As it closely adheres to the mucous membrane of the stomach, the powder may be found in the dead body, and separated by washing from the organic matter with which it is mixed. It is quite insoluble in water, and therefore may be procured as a sediment from organic liquids by washing and decantation. Strychnia may be obtained from it by a process described under that alkaloid (*post*); but, owing to the small proportion present, there will be some difficulty in procuring strychnia in a crystalline state, unless from twenty to thirty grains of the powder are obtained.

FIG. 69.



Hairs of nux vomica without the powder, magnified 70 diameters.

FIG. 70.



Hairs of nux vomica mixed with the powder, magnified 70 diameters.

In the Tissues.—Nux vomica bears the same relation to strychnia that opium does to morphia. It is by the strychnia absorbed from the powder as it lies on the mucous membrane of the stomach, that the life of a person is destroyed. Hence the poison for which we must seek in the tissues, is strychnia (see p. 684). It is a remarkable fact that, in no case of poisoning by nux vomica yet recorded, so far as I have been able to ascertain, has strychnia been found in the blood or tissues of persons poisoned by it. There is no doubt that strychnia is absorbed from the powder and carried into the blood, where it operates fatally, but the quantity so absorbed is too small to be revealed by the most delicate processes at present known. In cases of poisoning with nux vomica, we must rely chiefly upon the discovery of the powder in the fluids of the stomach. (See case by Dr. Stevenson, *ante*, p. 663.)

The alkaloids strychnia and brucia may be detected in it by the following process: Digest the powder in a small quantity of diluted sulphuric acid by a water-bath heat. The substance should be well stirred with the diluted acid, which, after a short time, completely carbonizes it. The mass is heated to dryness, then treated with a small quantity of distilled water and filtered, by which an acid liquid of a pale sherry color is obtained. On neutralizing this liquid with potash or ammonia, and agitating it with two volumes of ether, the strychnia is separated, and may be obtained crystallized by the evaporation of the ethereal solution. (See p. 688, *post.*) The strychnia may also be obtained by dialysis. Ten grains of nux vomica, equal to $\frac{1}{10}$th grain of strychnia, gave satisfactory results. Prismatic crystals were procured which gave the appropriate reactions with the color tests. Brucia was also detected by the action of nitric acid on the crystals. In cases of poisoning by nux vomica, brucia should be detected as well as strychnia.

NUX VOMICA BARK.

This was formerly confounded with the Cusparia or Angostura bark, and has been long known under the name of false angostura. It contains strychnia and brucia, the latter in large proportion, and in its effects on the body, whether in the state of bark, infusion, or decoction, it resembles the seed of nux vomica. About the latter end of the last century, a quantity of this bark was distributed over Europe, mixed with the angostura or cusparia, and numerous fatal accidents occurred before the true nature of the poisonous bark was discovered. Dr. Husemann has given a full account of these cases in Reil's *Journal für Toxikologie*, 1857, H. 4, p. 511. He has collected eleven cases of poisoning by this bark.

The Strychnos colubrina, or Snakewood of the East Indies, is supposed by some to be the wood of the nux vomica, and to produce similar effects. The term Snakewood is applied, in the East, to a variety of woods which, when made into cups, impart a bitter taste to water; and the water is then considered to be an antidote to the bites of venomous serpents.

The nux vomica bark is now seldom met with. It is known by the fracture of the bark acquiring a strong red color on being touched with nitric acid. From its infusion or decoction both strychnia and brucia may be obtained.

ST. IGNATIUS'S BEANS.

These are the seeds of the *Strychnos ignatii* or the *Ignatia amara* (Cabalonga). The tree producing them is said to abound in the Philippine Islands. According to Pereira the fruit has a pyriform shape, and contains twenty of the seeds. They were first made known by the Jesuits, and they were named after their patron. Pelletier and Caventou found that they contained 1.2 per cent. of strychnia; when taken in powder, they produce symptoms and effects similar to those caused by strychnia. They were formerly used as a febrifuge in medicine. A case is related by Husemann, in which a man, æt. 40, took one-half of a bean in brandy to cure an attack of fever. He suffered from the usual tetanic symptoms, and narrowly escaped with his life. Four other cases are referred to by this writer. (Reil's *Jour. für Tox.*, 1857, H. 4, p. 520.) Mr. Bennett gave half a drachm of the seed, procured at Manilla, to a dog. In twenty-five minutes the dog was suddenly seized with tetanic convulsions. There was panting respiration, with trembling of the muscles, and twitchings of the face, while frothy saliva issued from the mouth. There was a remission in five minutes. The animal was conscious. In a quarter of an hour after the first access of the symptoms, there was a general convulsion of the body, under which death took place. No particular appearances were found. The stomach had a pinkish hue, and the powdered seed was found in it. There was congestion of the liver, and the blood was generally liquid. In a similar experiment on a dog, with the same quantity of seed, the tetanic symptoms came on quite suddenly in half an hour, and after various attacks the animal died, in three-quarters of an hour from the time of seizure. (*Lancet*, Aug. 31, 1850, p. 259; and Braithwaite's *Retrospect*, 1850, p. 415.) Brucia is associated with strychnia in these beans, and according to some it is the predominating alkaloid. (*Pharm. Jour.*, Feb. 1875, p. 661.)

STRYCHNOS (UPAS) TIEUTE—(JAVA POISON).

The plant which yields this variety of Upas poison is the *Strychnos tieute.* It is described as a large climbing shrub, which grows in Java, and is known under the name of *Tshettik.* The extract contains strychnia, but no brucia. Its effects are similar to those of nux vomica. It produces tetanus, asphyxia, and death.

BRUCIA.

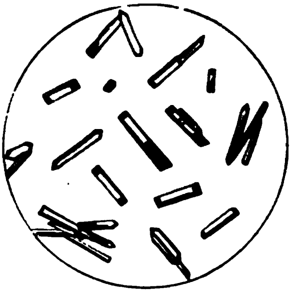
This is an alkaloid generally associated with strychnia. The seeds of the nux vomica yield chiefly strychnia, while the bark of the tree is said to contain brucia in larger proportion than strychnia. It derives its name from Bruce, the Abyssinian traveller, as it was extracted from the bark of a tree supposed to have been discovered by Bruce in Abyssinia, whereas the bark turned out to be that of the nux vomica from the East Indies. Brucia is also found in St. Ignatius's beans.

Symptoms and Effects.—This alkaloid and its salts produce in man and animals symptoms similar to those caused by strychnia. It is not so powerful a poison, and requires to be given in much larger doses. Magendie regarded it as having one-twelfth of the strength of strychnia, while Andral assigns to it one-sixth of the power. The latter view is more correct. As a medicine it may be given in doses of half a grain, a quantity which would prove fatal if strychnia were employed. Poisoning by brucia is rare. Casper refers to three cases of death from rat poison containing arsenic and brucia. No trace of brucia was found in the stomach. (*Ger. Med.*, 1857, p. 444.)

From a case of poisoning with this alkaloid, which occurred to Dr. Edwards, of Liverpool, it is necessary to give a caution to medical men respecting the possible criminal use of brucia. The symptoms which it causes so closely resemble those of poisoning with strychnia that, in the event of death, the latter poison only might be sought for and not found. The tetanic symptoms are more slowly produced, and the poison is not so rapidly fatal as strychnia; but these conditions may be altered by the larger quantity given. Hence, when in a suspected case, the color tests for strychnia fail to show the presence of this alkaloid, nitric acid should be added to the crystalline residue obtained, by the use of ether or chloroformic ether. (See p. 688.) The intense reddening produced by this test, with the other characters above mentioned, will indicate the presence of brucia. Although it somewhat resembles morphia in the action of nitric acid, it is easily distinguished from this alkaloid by iodic acid, which is not decomposed by it. The bitter taste of this poison is well marked, and is very persistent. Owing to an accident, a portion of brucia in very fine powder entered my eyes, mouth, and nostrils. The sense of bitterness of taste was immediately perceptible, and it did not disappear until after the lapse of more than two hours.

Analysis.—Brucia is much more soluble in water and alcohol than strychnia, and its solutions have an intensely bitter and persistent taste. It may be separated from strychnia by alcohol. Its hot aqueous solution has a strong alkaline reaction. It is easily dissolved by diluted acids, and forms crystallizable salts. Among these the sulphate is well marked by its crystallization in prisms, which are larger and longer than those of the sulphate of strychnia, and they are truncated at the ends. (Fig. 71.) 1. By dissolving it in hydrochloric acid, and adding ammonia, it may be obtained in groups of stellated crystals. 2. Sulphocyanide of potassium separates it from its solutions in crystalline tufts. 3. Nitric acid gives to brucia and its salts, either solid or in solution, a deep blood-red color. 4. If the liquid thus reddened be gently warmed, and chloride of tin is gradually added to it when cold, it assumes a deep crimson color. An excess of the chloride of tin or nitric acid will destroy this color. These changes of color are well observed on a plate of opal glass. 5. Hydrochloric and iodic acids dissolve it without change. 6. Strong sulphuric acid colors brucia of a rich rose-pink tint; on adding to this mixture bichromate of potash, oxide of manganese, ferricyanide of potassium, or peroxide of lead, the blue, violet, and purple colors observed in experimenting on strychnia are *not* produced. The mixture slowly acquires an olive or greenish-brown color. 7. Sulphomolybdic acid, which produces slowly a pale blue with strychnia, gives with brucia and its salts a red color passing to a deep maroon, and ultimately to a blue-black. 8. Chromate of potash does not act upon a solution of brucia as upon that of strychnia. The solutions of the salts of brucia are precipitated by potash and other alkalies.

Fig. 71.



Crystals of sulphate of brucia magnified 124 diameters.

AKAZGA.

This is an ordeal poison of the West Coast of Africa which belongs to the strychnos species. It has been brought to the notice of the profession by Dr. T. Fraser, who has published a full description of the plant which yields it. (*On the Character of the Akazga Plant*, 1867.) The seed has a globular form with somewhat flattened sides, and is almost from half to three-quarters of an inch in diameter. Its external surface is covered with a downy layer of long hairs, but it does not present the velvety appearance of the seeds of nux vomica. The seed is bitter, and the bark is strongly so. As used in the African ordeal for the detection of witchcraft, it causes death by tetanic convulsions like strychnia. Dr. Fraser procured from it an alkaloid resembling strychnia (*akazgia*). This, he states, agrees with strychnia in its physiological effects, and in producing the same color reactions with the color tests. It is not readily obtained in a crystalline form.

CHAPTER LXIX.

STRYCHNIA AND ITS SALTS—SYMPTOMS—CHRONIC POISONING—APPEARANCES AFTER DEATH—FATAL DOSE—PERIOD AT WHICH DEATH TAKES PLACE—VERMIN AND INSECT KILLERS—TREATMENT OF POISONING WITH STRYCHNIA—HYPODERMIC INJECTIONS.

STRYCHNIA AND ITS SALTS.

STRYCHNIA (from *ὁ στρύχνος*, a term applied to plants supposed to have the narcotic properties of nightshade) is an alkaloid extracted from nux vomica. It was discovered in 1818, but it was not until eight or ten years after its discovery that it came into medicinal use. Its properties as a deadly poison have been long known to medical men, but they have only within the last twenty years been brought prominently before the public, and this alkaloid has now acquired a fatal notoriety. From the time of its discovery up to 1856 it had caused at least *seventeen* deaths, and numerous cases of accidental poisoning, from the effects of which the persons had recovered. Husemann in 1857 had collected thirty-five cases of poisoning by strychnia and its salts; namely, twenty-four with the pure alkaloid, eight with the nitrate, two with the sulphate, and one with the acetate. (Reil's *Journal*, 4 H., p. 521, 1857.) A large amount of experience has now been accumulated on this form of poisoning.

Symptoms.—When strychnia is taken in solution, it has a warm and intensely bitter *taste*. This, of course, is not necessarily perceived when it is swallowed in the form of a pill. At an interval varying from a few minutes to one hour or longer, and sometimes without any premonitory symptoms, the person is suddenly seized with a feeling of suffocation and great difficulty of breathing. He is restless and uneasy, and complains of a feeling of choking or impending suffocation. There are twitchings and jerkings of the head and limbs—a shuddering or trembling of the whole frame. Tetanic convulsions then commence suddenly with great violence, and nearly all the muscles of the body are simultaneously affected. The limbs are stretched out, the hands are clenched; the head, after some convulsive jerkings, is bent backwards, the whole body is as stiff as a board, and assumes, by increase of the convulsions, a bow-like form (opisthotonos), being arched in the back and resting on the head and heels. During the fit, the head is firmly bent backwards, and the soles of the feet are incurvated or arched and everted. The abdomen is hard and tense; the chest is spasmodically fixed, so that respiration appears to be arrested; the face assumes a dusky, livid, or congested appearance, with a drawn, wild, or anxious aspect, the eyeballs are prominent and staring, and the lips are livid. The features have been observed to assume in some cases the peculiar appearance given by the sardonic grin (*risus sardonicus*). The patient complains of a choking sensation with thirst and dryness of the throat. An attempt to drink is often accompanied with a spasmodic closure of the jaws, by which the glass or vessel is broken or bitten. In several cases of poisoning by strychnia there has been from the outset a sense of impending dissolution, and one of the first exclamations made by the patient has been, "I shall die." The intellect is generally clear and unclouded during the intervals of the paroxysms, and the patient appears to have a full sense of his danger. After a succession of fits, and generally shortly before death, there may be a loss of consciousness. This was observed in a case which occurred to Dr. Ogston, and in that of Mrs. Dove. Pain is occasionally felt at the pit of the stomach, and during the paroxysms there is severe suffering from the violent spasm of the voluntary muscles. The consciousness of the access of the fit is very remarkable. The patient calls out loudly, "It is coming," and screams or shrieks, asking at the same time to be held. He in vain seeks for relief in gasping for air and in requiring to be turned over, moved, or held. Sometimes there is frothing at the mouth, and this froth is bloody from injury to the tongue. With respect to the muscles of the lower jaw, these, which are the first to be affected in tetanus from disease, are generally the last to be affected by this poison. The jaw is not primarily attacked, and is not always fixed during the paroxysm. It is relaxed in the interval, and the patient can frequently speak and swallow. When the jaw has been fixed by spasm, unlike the lockjaw of disease, this has come on suddenly in full intensity, with tetanic spasms in other parts, and there are intermissions which are not commonly witnessed in the tetanus of disease (see p. 103). The sudden and universal convulsion affecting the voluntary muscles has been sometimes so violent that the patient has been raised up and even jerked off the bed. During the convulsions the pulse is very quick. After an interval of half a minute to one or two minutes, the convulsions subside; there is an intermission; the patient feels exhausted, and is sometimes bathed in perspiration. Dr. Falck, who has made a most minute examination of the action of this poison on animals, affirms that it is an error to assert that the convulsions are always of a tetanic character. Clonic convulsions are also met with, and some described as tetanic are of this character, the pauses between the spasms being so slight as to escape notice. (*Vierteljahrs.*, 1874, vol. 2, p. 45; also vol. 1, p. 103.) It has been noticed that the pupils were dilated during the paroxysms, while in the intermission they were contracted. (See *Brit. Med. Jour.*, May 2, 1874, p. 577.) Slight causes, such as the attempt to move, or a sudden disturbance, or even touching the persons lightly, will frequently bring on a recurrence of the convulsions. In cases likely to prove fatal, they rapidly succeed each other and increase in severity and duration until at length the patient dies exhausted. The tetanic symptoms produced by strychnia, when once clearly established, progress rapidly either to death or recovery. The patient is conscious, and the mind is commonly clear to the last. He has a strong apprehension of death. The duration of the case, when the symptoms have set in, is reckoned by minutes, while in the tetanus of disease, when fatal, it is reckoned by hours, days, or even weeks. As a general statement of the course of these cases of poisoning, within *two hours* from the commencement of the symptoms the person either dies or recovers, according to the number and severity of the paroxysms and the strength of his constitution. Death sometimes takes place in a paroxysm. (See case by Mr. Lawrence, *Lancet*, June, 1861, p. 572.)

The *time at which the symptoms commence* appears from the recorded cases to be subject to great variation. In poisoning by nux vomica the symptoms are generally more slow in appearing than in poisoning by strychnia. Until they set in suddenly, the patient is capable of walking, talking, and going through his or her usual occupations. On an average, in poisoning by strychnia, the symptoms appear in from five to twenty minutes. The interval which may elapse between the taking of the poison and the first appearance of symptoms, has formed a subject of discussion, and in the case of the notorious criminal, Palmer, it was made a cardinal point of the medical defence. Dr. Warner, æt. 39, took by mistake half a grain of sulphate of strychnia; the symptoms began, in less than *five minutes*, by constriction of the throat, tightness of the chest, and rigidity of the muscles on attempting to move. He first complained of want of air, and requested the windows to be opened. He died in from fourteen to twenty minutes, his mind remaining clear until the last. (*Brit. Amer. Journal*, August, 1847.) In that of Mrs. S. Smyth, of Romsey, three grains of strychnia were taken by mistake for salicin. This lady was in violent spasms in from *five* to ten minutes afterwards, and she died in one hour and a quarter. (*Pharm. Journal*, 1848, vol. 2, p. 298.) In another case convulsions came on in five minutes. (*Ann. d'Hyg.*, 1861, vol. 1, p. 133.) A girl, æt. 13, took one grain and a half of strychnia in solution on an empty stomach; the symptoms began by twitchings of the muscles rather more than *an hour* after the poison was taken; and she died in a violent tetanic fit in two hours and a half after she had taken the poison. (Mr. Bennett, in *Lancet*, August 31st, 1850.) This was a case in which, according to theory, the symptoms should have commenced within a few minutes! I have elsewhere referred to the case of Assistant-Surgeon Bond (p. 106). This gentleman took two pills containing two grains of strychnia, at 11.30 P.M., believing at the time that he had taken two aperient pills. He went to bed, and was soon sound asleep. About 1.30 A.M., *two hours* after taking the pills, he started from his sleep, awoke his wife, and said that he should die. Convulsive movements of the limbs with difficulty of breathing followed immediately. At 2.20 A.M. he was seen by my informant, and was then suffering from tetanic convulsions. No suspicion existed in the mind of deceased, or of any one about him, that he had by mistake taken poison. He was bled, when a severe convulsion came on suddenly, and he died about three hours after he had taken the pills. This case occurred in December, 1857, and a report was forwarded to me in March, 1858. In a case reported by Dr. Ogilvie, of Alexandria, in which about four grains of strychnia were taken, the symptoms did not appear until *an hour* after the poison was taken. (*Med. Times and Gaz.*, Oct. 30th, 1858, p. 443.) In another instance there was a similar interval. (*Lancet*, Aug. 31, 1850.) Drs. Lawrie and Cowan have reported the case of a medical man who, in June, 1853, took three grains of strychnia, dissolved in rectified spirit and diluted sulphuric acid. He went to bed and slept for about *one hour and a half*, when he awoke in a spasm, uttering loud cries which alarmed the household. Under treatment this gentleman recovered. (*Glasgow Med. Journal*, part 14, July, 1856.) Dr. Anderson met with a case, in which *two hours and a half* elapsed before the appearance of symptoms. In this case the man took three and a half grains of strychnia by mistake for muriate of morphia. He recovered. (*Ed. Monthly Journal*, 1848, p. 566.) The longest interval recorded was in the following case: A boy, æt. 12, swallowed a pill containing three grains of strychnia. No symptoms appeared for *three hours;* they then set in, in the usual way, and death took place in ten minutes. It was clearly proved that the pill taken contained three grains of strychnia with mucilage. The pills had been prepared eight months previously for the purpose of poisoning dogs; hence they were hard, and underwent only slow solution in the body. (*Lancet*, 1861, vol. 2, p. 480.) Other cases are reported in which the symptoms appeared in from ten minutes to three-quarters of an hour. (*Guy's Hosp. Reports*, Oct. 1856, p. 346; also *Reil's Journal für Toxikologie*, 1857, vol. 2 H., p. 499.) On an average, it may be stated that symptoms usually appear in from five to twenty minutes.

The form in which the poison is administered or applied has a considerable influence on the time at which the symptoms commence. Thus when strychnia is given in pills, especially if, as in the above case, they are hard, the symptoms are much longer in appearing than when the poison is taken in solution. Mr. Savory gave to a dog two bread pills, each containing one-quarter of a grain of strychnia. No symptoms of poisoning had occurred at the end of *two hours*, but the animal was found dead a short time afterwards. When strychnia was given in solution, the symptoms soon appeared, and death took place rapidly. (*Lancet*, 1863, vol. 1, pp. 515, 548.) It is remarkable that so simple a fact connected with the absorption of this poison, should have been wholly ignored by some of the experts who appeared for the defence of William Palmer (*Reg.* v. *Palmer*, C. C. C., 1856). Palmer gave to the deceased, Cook, two pills containing strychnia. No symptoms were observed for fifty-five minutes. More than one expert swore strongly that this interval rendered it impossible that the symptoms could have been caused by strychnia! It was fortunate for the ends of justice that the jury put no confidence in strong statements thus made on oath without any reasonable amount of experience to warrant them. The cases above mentioned will show the great danger of trusting to such dogmatic opinions.

If the poison is applied hypodermically to the cellular membrane, to an ulcerated or diseased surface, or even a healthy mucous surface, absorption takes place rapidly, and the interval for the production of symptoms is proportionably short. Dr. Schuler relates a case of amaurosis in which about the twelfth part of a grain of strychnia was introduced into the punctum lachrymale at the corner of the eye. Three or four minutes had not elapsed when symptoms of poisoning appeared. There was convulsive respiration, with violent tetanic shocks, and the patient appeared about to die; however, these symptoms passed off, and he recovered. (*Med. Times and Gazette*, July, 1861.)

Another fact connected with the symptoms worthy of notice is, that there is a great exaltation of sensibility, and sometimes of the senses of sight and hearing; hence a slight touch may induce a tetanic paroxysm. On the other hand, patients suffering from the effects of strychnia have frequently derived great relief from being held, moved, restrained, or rubbed during the convulsive fit. In Dr. Lawrie's case (*supra*) great relief was given by the forcible extension of the body; and in the cases of Mrs. S. Smyth (*supra*) and J. P. Cook (*post*) each desired to be turned over. In fact, while a slight touch, by acting more as an excitant, may induce a spasm, a firm grasp has not produced this effect. (*Report on Strychnia*, by Dr. Steiner, Philadelphia, 1856, p. 14.) At any rate, a sense of relief has been experienced by the patient when held, moved, or rubbed; he has been able to swallow in the intervals of the fits, but at the same time to manifest, as in hydrophobia, a dread of the act of swallowing. The symptoms of poisoning by strychnia have been mistaken for those of tetanus. (For the means of distinction, see *ante*, p. 104.)

The *duration* of the convulsive fit is subject to great variation. In some cases it has not exceeded half a minute; in others it has lasted eight minutes. On an average it has probably not exceeded two minutes. The number of fits has varied from two to seven or more. Patients have died after one or two fits, sometimes during the convulsive spasm, the intercostal muscles becoming fixed and thus leading to asphyxia, at others during the remission, and in this case death takes place from exhaustion. The length of the interval is in no two cases alike. The convulsions have subsided; but there has often remained difficulty of breathing, speaking, or swallowing. One fact, noticed in all the cases, has been the perfect consciousness of the patient during the intervals between the fits.

Chronic Poisoning—Accumulative Properties. — Some facts, elsewhere related, show that strychnia given medicinally does not appear to accumulate in the system. Any causes, however, which prevent or interfere with elimination may lead to the accumulation of the poison in the blood and a sudden accession of tetanic symptoms. In one case reported a slight increase of dose led to death. The late Dr. Pereira has recorded a similar case, which proved fatal from a sudden access of tetanus, although the medicinal doses had been borne with impunity for several days. (*Mat. Med.*, vol. 2, pt. 1, p. 664; see also on the effects of small medicinal doses, Andral, *Clinique Médicale*, by Spillan, 1836, p. 890.)

Appearances.—Externally the body is in general relaxed at the time of death, but it soon stiffens, and the muscles retain an unusual rigidity for a long period. The hands are clenched, and the feet arched or turned inwards. In the case of J. P. Cook, the rigidity of the body and limbs is stated to have been well marked on exhumation after two months' interment. In some instances, when death takes place in a spasm, the rigidity may continue, and maintain the body in the attitude given to it by the spasm. This occurred in a case related below, in which the opisthotonic condition was retained after death, unless we assume that this was the result of a post-mortem action of the muscles, which is not probable. The late Dr. Geoghegan observed in one case that the tetanic spasm was merged in the subsequent rigor mortis. (*Dub. Med. Press*, June 25, 1856, p. 404.) It by no means follows, however, that the dead body should always be found in an attitude indicative of convulsions. It may be found relaxed, or the only signs of the past existence of convulsions may be a clenched state of the hands, a separation of the legs, and an arched condition of the soles of the feet. (See case *Med. Times and Gaz.*, Jan. 24, 1857, p. 96.) In two cases of suicide by strychnia, which occurred in Soho in October, 1872, one of the deceased, a woman, was found sitting in an arm-chair at a table, her hands crossed, and a Bible before her. Her face was livid, and her body rigid. The man was found lying on the floor, his hands crossed on the chest. A large quantity of strychnia was found in the stomachs of both.

In the cases of Mrs. Vyse's two children (*Reg.* v. *Vyse*, C. C. C., 1862), who died in less than an hour from the effects of Battle's vermin-killer, administered by the mother, Mr. Savory made the following observations. He saw the bodies soon after death. They were much discolored, livid, and although quite warm, were perfectly rigid. The younger, aged five years, was rigid all over; the elder principally about the jaws and neighboring parts. The rigidity gradually disappeared, and after twenty-four hours there was scarcely any indication of it remaining in the elder child. Decomposition had commenced, the front of the abdomen presenting a green discoloration. The body of a person poisoned by strychnia may therefore be found in a non-rigid state within the ordinary period after death; but in most recent cases, it is not unusual to find the hands clenched, and the feet arched or turned inwards—incurvated. In some instances of undoubted strychnia-poisoning no particular degree of rigidity has been found at any period after death.

In rabbits poisoned by similar doses of strychnia, I have observed the body of one to remain perfectly rigid for a week, while another had lost all rigidity, and had begun to putrefy after thirty-six hours. The circumstances which affect the commencement and duration of this condition of the dead body have been elsewhere described (*Prin. and Pr. of Med. Jur.*, vol. 1, p. 53). The experiments of Brown-Séquard have conclusively shown that it is not any special influence of the poison on the muscles, but the mode in which it operates on the system, that determines the commencement and duration of rigidity in the dead body.

In an accurately observed case recorded by the late Professor Casper, of Berlin, the body was examined forty-one hours after death. It presented the slight greenish tinge of incipient putrefaction in the loins; there was slight humidity; the expression of the face was that of one quietly sleeping—the eyes were closed, the pupils were neither contracted nor dilated. Rigidity was present in its usual degree for the time of observation, well-marked as it always is in the masseter muscles, by which the jaws were firmly closed, and more strongly marked in the limbs, which were lying parallel with the trunk. The feet were not incurvated; the fingers, as in other dead bodies, were half flexed inwards, and the nails were blne. There was no evidence of tetanic, still less of opisthotonic stiffness or rigidity of the body. In short, this body was externally precisely like a thousand other bodies ("äusserlich genau wie tausend andre Leichen") which had come before him; and any physician not informed of the mode of death, would have no suspicion whatever of death from strychnia from the *external* appearances. (See report of this remarkable case revised by Casper within a few hours of his own death, in Horn's *Vierteljahrsschrift für gerichtliche Medicin*, Juli, 1874, p. 7.) A man who clears away an error in medical jurisprudence, does as much service to science, as he who discovers a new truth. Another remarkable external appearance which may be here noticed is, a greater or less lividity about the head, body, and limbs, with fixedness of the joints.

Among the *internal* appearances are—congestion of the membranes and substance of the brain, as also of the upper part of the spinal marrow; congestion of the lungs and air-passages; the heart is contracted and empty, but its right cavities, in other instances, have been found distended with liquid blood. The blood is dark-colored and liquid throughout the body. The mucous membrane of the stomach and intestines has occasionally presented patches of ecchymosis or congestion, probably depending on extraneous causes, since, in other instances, these parts have been found quite healthy. The bladder is generally empty.

A gentleman who had taken about six grains of strychnia was found dead. The body was examined on the same day. The face was pale, the features were calm and placid, the eyes were closed, the pupils natural. The arms, although still warm, were rigid and bent at right angles, lying across the chest. The whole body was rigid and curved in a state of opisthotonos, resting upon the heels and back of the head. There was some lividity on the right side. The feet were slightly turned inwards. The muscles were of a bright red color, the lungs were slightly congested, the heart was larger than natural, the right cavities were distended with dark fluid blood, the left cavities contained but a small quantity. The stomach was congested, the mucous membrane presenting some dark-brown patches. It contained about six ounces of food, and in some of the folds strychnia was visible. The liver, gall-bladder, kidneys, spleen, and intestines presented nothing unusual. The bladder was in this case nearly full of urine. The brain was slightly congested. (Mr. Porter, in *Dublin Hosp. Gaz.*, Aug. 1, 1858, p. 227; see also paper by Dr. Geoghegan, *Dublin Med. Press*, June 25, 1856, p. 401.)

In a case in which a person died in six hours from a dose of three grains, the rigidity of the body seven hours after death was so great as to allow it to be lifted by the heels. It was as stiff as wood, and all the muscles were firmly contracted. Thirty-six hours after death this rigidity had diminished except in the fingers. The lungs were congested; the heart was flabby, the right cavities containing dark-colored blood, partly fluid and partly coagulated. The liquid portion appeared to be full of air-bubbles. The only appearance observed in the abdomen was congestion of the kidneys. In the head, the membranes of the brain (dura and pia mater) were congested; the substance of the brain was also congested. The ventricles contained much serum; the choroid plexus was congested and of a dusky color. The upper part of the spinal marrow was very red superficially, and the canal appeared to be full of serum. The scalp was loaded with blood. (*Guy's Hosp. Reports*, Oct. 1857, p. 484.) Mr. Wilkins forwarded to me the stomach of the deceased. The mucous membrane was very rugose, and of a dark brownish-red color. At the greater end there was a red patch, arising from congestion, covering about three-quarters of an inch of the mucous surface. There was a diffused redness of the lining membrane, amounting to deep lividity at the two ends of the stomach. It was softened, and a thick layer of mucus adhered to it. It contained about seven ounces of fluid of a light reddish-brown color. This was removed and reserved for analysis. In a case that occurred to Dr. Ogston, in which a man died from three-quarters of a grain in about three-quarters of an hour, the appearances were similar, except that in this short period the congestion was much greater. The mucous membrane of the stomach was of a dark-red color from intense congestion, and a thin layer of blood adhered to it. The duodenum and jejunum were also reddened. The veins of the spinal cord and its sheath were congested. (*Lancet*, April 19th, 1856, p. 428; see also *Med. Times and Gaz.*, 1854, Dec. 16th, p. 924.) In a case reported by Mr. Startin, a man who had taken strychnia medicinally died in less than three hours from a dose of a grain and a half. On inspection, there were extensive patches of extravasated blood beneath the arachnoid membrane of the lower half of the spinal cord. (*Med. Times and Gaz.*, March 21st, 1857, p. 297.)

Of the appearances produced in poisoning by strychnia, there are none which can be considered strictly characteristic. Congestion of the membranes of the brain and spinal marrow is probably the most common. With regard to the state of the heart and lungs, their condition, as to fulness or emptiness, must depend rather on the mode of dying than on the actual cause producing death. The condition of the *heart* in these cases requires a brief notice. It has been incorrectly assumed, from experiments on animals, that in death from strychnia the right cavities of the heart are invariably distended with blood, and a further erroneous deduction has been made, that, if the heart has been found empty in any case, this condition is inconsistent with death from strychnia! In the *Guy's Hospital Reports* for Oct. 1856, p. 346, I have collected fifteen fatal cases of poisoning by strychnia, comprising, I believe, all that had been recorded up to that date, in which the details were known. Out of fifteen fatal cases, the body was inspected in ten; and in six of these inspections the heart was found either *empty*, sometimes contracted and sometimes flabby, or there was but little blood present. Further, these cases show that the condition of this organ, as to emptiness or fulness, does not depend on the fact whether the head has or has not been opened before the chest is examined.

In two cases, that of Greene, tried at the Chicago Circuit Court, and of Azenath Smith, tried in Canada, the heart was found healthy but empty in all its cavities. (*Poisoning by Strychnia*, p. 45.) In the case of Dr. Gardiner, who died in three hours and a half from the effects of strychnia, Dr. Steiner states, that while the membranes of the brain and upper part of the spinal marrow were congested with dark fluid blood, the heart was small, contracted, and contained no blood. (*Report on Strychnia*, 1856, p. 15.) Drs. Scholefield and Wright met with a case which proved fatal in about two hours, in which the heart was small, contracted, and nearly empty, and in this case the brain was not examined. (*Ed. Med. Jour.*, Nov. 1858, p. 410.)

These facts demonstrate that, in death from strychnia, the condition of the heart may vary, and that there is not the slightest ground for the assertion that emptiness of its cavities is inconsistent with death from this poison.

The state of the *lungs* and air-passages is liable to some variation. In the case of Mrs. Dove (*Reg.* v. *Dove*), York Summer Assizes, 1856, these organs were found highly congested. This lady died on the sixth day, after having had doses of strychnia administered to her at intervals during that period. In this case, forty-two hours after death, the muscles of the body were relaxed, but the limbs preserved some rigidity, the hands and feet being incurvated by muscular contraction. The membranes of the brain, especially the inner membrane (the pia mater), were much congested. There was bloody serum beneath this membrane and in the ventricles. The substance of the brain, as well as the membranes and substance of the spinal cord, were congested. The cavities of the heart were nearly empty; the small quantity of blood therein was dark and fluid. The blood was generally fluid, and of the consistency of treacle. The lungs and air-passages were engorged with dark blood, presenting the appearance of pulmonary apoplexy; the mucous membrane of the windpipe was of a dark plum color, and was covered on its surface with a dark-colored mucus. The other organs, including the stomach, were healthy. In the stomach there were slight appearances of congestion.

The late Professor Casper, of Berlin, who had had the largest medico-legal practice in Germany, states that out of nearly 1200 medico-legal inspections made by him up to December 10, 1863, no case of death from strychnia had come before him. At that date he made a careful examination of the body of a man, who had destroyed himself with strychnia, with a view, if possible, of fixing the special appearances produced by this poison, and of isolating them from those casual conditions of the dead body, which have been wrongly described as characteristic of the effects of strychnia. On December 10, 1863, a healthy man, æt. 30, swallowed at 5 o'clock P.M., from five to six grains of strychnia. For about an hour, he lay in his room quietly. At this time spasms commenced, and in his attempt to reach a window he fell, and lost all power of moving his legs. He was not seen for another hour, when he was found lying on the floor and asking for water. In attempting to raise himself, he was seized with tetanic convulsions affecting the whole of his muscles; he had three of these fits in a severe form, and died in the last at 8.15 P.M. During the spasms, as well as in the intervals, there was complete consciousness.

The *external* appearances in this case have been already described (p. 677). The two outer membranes of the brain were filled with blood, which throughout the body was generally fluid as in death from asphyxia; it was of a light reddish color, as in poisoning by carbonic oxide or prussic acid. The brain and spinal marrow were healthy. The muscles of the throat and gullet were of a dark violet color, unlike the other muscles of the body. The lungs were natural; not congested. The right cavities of the heart were collapsed and empty, and the left cavities contained but little blood. The large vessels were also nearly empty. The spleen was congested. The stomach was half full of a mass of partly digested food; the mucous membrane was pale, firm, and softened, and, when minutely examined by a lens, was found to be perfectly natural. The mucous membrane of the whole of the intestinal canal was in the same healthy state. The kidneys were healthy, and not congested. The spinal marrow was especially examined throughout its whole extent, as well as the roots of the spinal nerves. It was cut into in various directions; and in no part did it present any appearance deviating from the healthy condition. So far as appearances went, there was no visible cause of death in the case of this adult healthy man dying in less than four hours from a large dose of this powerful poison, and obviously from its immediate effects. In this respect, strychnia resembles other alkaloidal poisons. (Horn's *Vierteljahrsschrift*, Juli, 1864, p. 28.)

Casper considers the peculiar color of the muscles of the throat and gullet as worthy of notice. This was the only deviation from the ordinary appearances which he had been accustomed to meet with in cases of violent death. If he had had no previous experience of the condition of the body in death from strychnia, he had had, during a long and active life, unsurpassed opportunities of observing the appearances in all other kinds of violent death. He was thus in a better condition than others, to fix upon any that were really characteristic of poisoning by strychnia. Although the examination of a dead body is thus proved to throw but little light upon the question of death from strychnia, still a medical jurist has in the symptoms, and in their mode of occurrence and progress, sufficient data for a safe opinion.

Fatal Dose—Quantity required to Destroy Life.—The medicinal dose of strychnia for an adult ranges from $\frac{1}{30}$th to $\frac{1}{12}$th of a grain. The $\frac{1}{16}$th of a grain is an average dose. This quantity has, however, operated as a poison on a child. It caused the death of a child between two and three years of age, in four hours. In a case reported by Dr. Danvin, three-quarters of a grain killed a child, æt. 7½, in half an hour. (*Annales d'Hygiène*, 1861, vol. 1, p. 133.) In two cases of adults, in each of which a quarter of a grain had been taken by mistake, the patients recovered only under early treatment. (*Lancet*, July 27, 1856, pp. 107, 117.) The smallest fatal dose in an adult was in the case of Dr. Warner. *Half a grain* of the sulphate of strychnia here destroyed life. (*On Poisoning by Strychnia*, pp. 138, 139.) In another instance a woman, æt. 22, an in-patient of the Jersey Hospital, took by mistake four pills containing in each one-eighth of a grain of strychnia. Symptoms soon appeared, and this dose (half a grain) proved fatal. (*Dub. Med. Press*, Sept. 17, 1852, p. 182.) So powerful are the effects of this drug in certain cases, that ordinary medicinal doses can scarcely be borne. A gentleman took one-twentieth of a grain of strychnia in six doses during a period of two or three days. Several fits of tetanus occurred, although half a grain had not been taken altogether. It is probable in such cases that elimination is either arrested or imperfectly performed. In May, 1859, Dr. Tweedie informed me of a case in which he had prescribed for a gentleman pills, each containing $\frac{1}{15}$th of a grain of strychnia. He took altogether five of them, or *one-third* of a grain, at proper intervals. The patient was seized with the most alarming tetanic convulsions, continuing for some time. There was also opisthotonos of a severe kind. He only slowly recovered. A *fatal dose* of strychnia for an adult may be assigned at from half a grain to two grains.

As in other cases of poisoning, many recoveries have taken place, even after large doses of strychnia have been taken. There are at least three instances on record in which persons have recovered after taking one grain. A case of recovery from two to three grains is reported in the *Lancet* for 1861, vol. 2, p. 169. In the same journal for 1863, vol. 1, p. 54, Dr. Angell describes a case in which a girl recovered in six or seven hours from a dose of *four grains* of strychnia. When first seen, she was sensible, and while talking was suddenly seized with the usual tetanic symptoms—opisthotonos, concave contraction of the hands and feet, the muscles rigid, the eyes natural, the pulsations of the heart considerably increased, the respiration difficult, and a great fear of death. She had only three paroxysms, and to this probably her recovery was due, as her system was not exhausted by severe and frequent convulsive attacks. There is one instance reported in which a person is said to have recovered from a dose of seven grains of strychnia. (*Med. Gaz.*, vol. 41, p. 305.) In reference to this alleged recovery from large doses, it may be a question whether the strychnia was not mixed with some other substance, whereby its poisonous properties were weakened. A fatal dose for an adult may be assigned at from *half a grain to two grains*. Instances of recovery from doses above one or two grains must be regarded as exceptional.

Period at which Death takes place.—In fatal cases death generally takes place within two hours after the taking of the strychnia. One of the most rapidly fatal cases recorded is that of Dr. Warner (p. 681). The symptoms commenced in five minutes, and he was dead in *twenty minutes*. In a case privately communicated to me, ten grains of strychnia, given by mistake for sulphate of quinine, killed a patient in *ten minutes*. In Dr. Ogston's case, in which three-quarters of a grain was taken, the man appears to have died in less than a quarter of an hour from the commencement of the symptoms, and probably three-quarters of an hour from the time of taking the poison. (*Lancet*, April 19, 1856, p. 428.) In 1870 two deaths are reported to have occurred at Ypres, in Belgium, in which strychnia proved more rapidly fatal than in the preceding cases. M. Merghelynk took in pills, seven grains and a half of what he supposed to be hydrochlorate of quinine. Violent convulsions came on, and he died in a quarter of an hour. His wife, not suspecting anything wrong, took a similar dose, and died in *ten minutes*. A pill containing a grain and a half was given to a dog, which soon died under the usual symptoms of poisoning by strychnia. The supposed hydrochlorate of quinine was then examined, and it was found to be largely mixed with strychnia. The case of Madame Merghelynk is, with one exception, the most rapid on record. The late Dr. St. Clair Gray refers to a case which proved fatal in *five minutes*. (*On Strychnia*, 1872, p. 55.) In general, whatever may be the interval between the dose and first symptoms, death is rapid when the tetanic convulsions have once commenced. They may show themselves in a sudden and violent form, and the life of the patient then depends on the frequency and severity of the fits, and his age, strength, and constitution. In the case of the child that died from the sixteenth part of a grain (p. 680), the symptoms commenced in half an hour, but death did not take place for *four hours*. The protracted nature of this case may be explained by the smallness of the dose. (*Poisoning by Strychnia*, p. 138.) In the case of Dr. Gardiner, death took place in three hours and a half. (*Report*, by Dr. Steiner, 1856, p. 14.) In a case in which a gentleman took five grains of strychnia dissolved in orange-juice, by mistake for James's powder, the symptoms commenced in fifteen minutes, and death took place in *half an hour*. In the case of J. P. Cook, the poison was administered in two pills. There were no symptoms until after the lapse of fifty-five minutes, and death took place in from sixteen to twenty minutes after their commencement.

The longest duration of strychnia-poisoning was in a case communicated to me by Mr. Wilkins, of Newport, Isle of Wight. In February, 1857, a gentleman swallowed *three grains* of strychnia at 10 P.M. In three-quarters of an hour he was heard in his room groaning, and was there found in tetanic convulsions. They came on like shocks, affecting the whole muscular system, either spontaneously or on any attempt to speak, drink, or move. During the fits there was great congestion of the face, the pupils were dilated, and the eyes appeared starting from the sockets. The back was arched, the head thrown backward, and the feet were arched. He requested to be turned over. He was perfectly conscious, and held conversation in the intervals, and his skin was then bathed in perspiration. The pulse was 150, and he spoke in gasps. He could swallow, but with difficulty. The arms were involuntarily bent at the elbows, and the legs were stretched out; the twitchings of the face resembled those of epilepsy, but there was no frothing at the mouth. The heart continued to beat as in asphyxia after the cessation of respiration, the pulse gradually became more and more feeble, and ceased to beat at 4 A.M.—*i. e.*, *six hours* after he had taken the poison. (*Guy's Hospital Reports*, October, 1857, p. 483.)

Vermin and Insect-killers.—Although it is difficult to procure strychnia at a druggist's shop, it is extensively sold to the public by grocers, oilmen, and others, under the name of vermin-killers, in threepenny and sixpenny packets. *Butler's Vermin-killer* consists of a mixture of flour, soot, and strychnia. I have found the sixpenny packet to weigh about a drachm, and to contain from two to three grains of strychnia. As the poison is mechanichally mixed with the other ingredients, and is probably manufactured on a large scale, the proportion of strychnia in a powder is liable to variation. By the aid of a lens, the poison may be sometimes seen scattered in white particles through the colored powder. The threepenny packet contains about half the quantity of strychnia, but, as it will be seen, quite sufficient to destroy the life of an adult. In place of soot Prussian blue is sometimes used as a coloring substance.

Battle's Vermin-killer is a powder similar to that of Butler, containing a fatal proportion of strychnia, is also sold in packets. These powders are a fertile source of poisoning either through accident or design; they are openly sold by ignorant people to others still more ignorant. In *Reg.* v. *Vamplew* (Lincoln Autumn Assizes, 1862), it was proved that the prisoner, a girl under 13 years of age (!), had purchased one of these powders at a village shop and had destroyed her master's infant with it. There was also reason to believe that this girl had destroyed two infants by similar means in two other families where she had acted as nurse. The children had all died suddenly in fits! In Vamplew's case I examined a similar powder purchased for threepence at the same shop, and found it to consist of about thirteen grains of flour, colored with Prussian blue and mixed with three-quarters of a grain of strychnia. Another Battle's powder, purchased in London for threepence, weighed, like this, about thirteen grains, and a sixpenny packet weighed twenty-three grains. The poison is, therefore, in a more concentrated form than in Butler's powder.

A case in which a young woman was killed in two hours by a threepenny packet of this powder was communicated to me in July, 1864; and another in which a drachm-packet of Butler's killer destroyed a girl, æt. 17, in one hour, has been reported by Mr. Saville. (*Med. Times and Gaz.*, Nov. 1857.) It would be easy to add to these many other fatal cases which have fallen within my own knowledge; but they present nothing out of the usual course. The persons have all died under the ordinary symptoms of poisoning by strychnia, in a well-marked form. The appearances in the body were similar to those seen in death from strychnia; but there is one caution to be given in reference to the examination of the stomach. As death is commonly rapid and there is no vomiting, the coloring matter, either soot or Prussian blue, should always be sought for in the stomach. Strychnia may or may not be found, according to the amount swallowed and the degree to which absorption has gone on during life. Some cases of recovery are reported. In 1859, a man recovered after taking a whole packet containing nearly three grains of strychnia (*Ed. Monthly Journal*, 1859, vol. 2, p. 407); and in 1860 Dr. Part met with an instance of recovery in which a girl took half a packet. In these exceptional cases the favorable results were probably due to vomiting excited by emetics. In 1863–7, over a period of five years, the deaths from strychnia in England and Wales were forty-one, and from vermin-killers, twenty.

Treatment.—If spasms have not already set in so as to close the jaws, we should, by the stomach-pump or by emetics, endeavor to remove the poison. In a case in which six grains of strychnia were taken, the life of the person appears to have been saved by the early use of the stomach-pump. (*Med. Times and Gaz.*, April, 1854, p. 316, and June 17, 1856, p. 809.) It has been supposed that emetics would not act in these cases; but this is an error (*Glasgow Med. Jour.*, July, 1856, part 14, p. 4). In several instances they have operated well and given relief. But the spasmodic symptoms may be sometimes so severe as to prevent the use of the stomach-pump or emetics. In this case the patient should be kept in perfect quiet and repose, so as to prevent as far as possible the recurrence of paroxysms. The senses are acutely impressionable. Any stimulus to them may cause tetanic convulsions. Thus light is intolerable, and the lowest whisper may be heard by the patient. A loud noise, the feeling of the pulse, or the sudden approach of a person to the bedside, has been known to bring on a paroxysm and lead to death by exhaustion.

Chloroform, chloral, and hypodermic injections of a variety of substances, such as morphia, physostigmia, curara, aconitina, conia, nicotina, and even bromide of potassium, are stated to have been used as physiological antidotes, with more or less success. In many of these cases the *post hoc* has no doubt been confounded with the *propter hoc*. On this mode of treatment, I must refer the reader to what has been elsewhere stated (*ante*, p. 64).

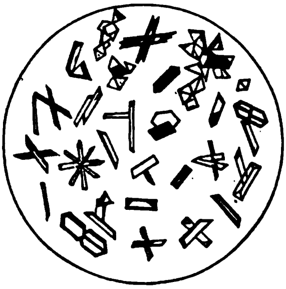
CHAPTER LXX.

POISONING WITH STRYCHNIA—CHEMICAL ANALYSIS—CRYSTALLINE FORM—TESTS IN THE SOLID STATE AND IN SOLUTION—DETECTION IN ORGANIC MIXTURES—IN THE TISSUES—DIALYSIS—NON-DETECTION IN THE DEAD BODY—QUANTITATIVE ANALYSIS—RECENT CASES OF MURDER BY STRYCHNIA—DETECTION OF STRYCHNIA IN VERMIN-KILLERS.

Chemical Analysis.—Strychnia is a white crystalline solid, scarcely soluble in water, but dissolved by rectified spirit, and in a smaller degree by ether. By the spontaneous evaporation of the alcoholic solution, the alkaloid is deposited in small well-formed crystals. It crystallizes in lengthened cuneiform octahedra, which have been described as four-sided prisms; also in flattened prisms, crossing each other at angles of 60°, bevelled at the ends. (See Fig. 72.) There are six or eight varieties of shape, so that too much importance must not be attached to this branch of the analysis. The crystals of delphinia resemble those of strychnia. As strychnia is separated from the solutions of its salts by the addition of ammonia it is usually deposited in long slender prisms. (See Fig. 73.)

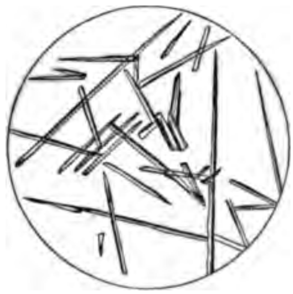
Tests for Strychnia in the Solid State.—The alkaloid strychnia possesses the following properties: 1. It is white, of an intensely bitter taste, even when it forms only $\frac{1}{30000}$th part of a solution. 2. When strongly heated on platinum, it melts and burns like a resin, with a black smoky flame; in a close tube it yields ammonia. 3. It is not perceptibly dissolved by cold water: it requires 7000 parts of cold and 2500 of boiling water for its solution. 4. It is dissolved by alcohol, benzole, ether, and chloroform, especially when in a recently precipitated state, and the last three liquids have the power of removing it from a non-acid watery solution. 5. It is easily dissolved by diluted acids, forming crystallizable and very soluble salts. From these acid solutions, unless too diluted, it is precipitated by potash or ammonia, in which strychnia is insoluble. 6. Strong nitric acid generally imparts to it a pale reddish color, owing to the presence of traces of brucia as an impurity. 7. Sulphuric acid produces no apparent change in it, but when to the mixture a small crystal of bichromate of potash, of ferricyanide of potassium, or a small quantity of finely powdered black oxide of manganese or of peroxide of lead is added, a series of beautiful colors will appear wherever the bichromate or the other substances meet the acid mixture. These colors commence with a deep sapphire-blue, passing rapidly through violet, purple, and crimson tints, until, by long exposure to the air, the mixture assumes a light amber-red color. Among these substances black oxide of manganese will be found preferable. By reason of its insolubility, it imparts no color to the liquid if strychnia is absent; and if the alkaloid is present, it brings out the colors more slowly, so that there is time to make a full observation of them. The hydrated or precipitated oxide of manganese may be used in place of the anhydrous compound. Permanganate of potash has

FIG. 72.



Various forms of crystals of strychnia, as they were obtained from an alcoholic solution, magnified 124 diameters.

FIG. 73.



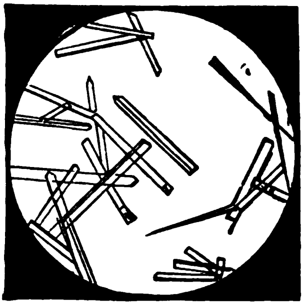
Crystals of strychnia obtained by adding ammonia to the sulphate, magnified 124 diameters.

been recommended as a substitute for the oxide, but it is objectionable on account of its solubility, and of its being already intensely colored. Dr. Letheby has suggested the use of a small galvanic battery for the production of the colored reaction. In this case sulphuric acid only is required. It presents no practical advantage over the use of oxide of manganese. 8. Sulphomolybdic and iodic acids produce no immediate change of color in strychnia, and thus clearly distinguish it from morphia.

The sulphomolybdic acid slowly imparts to strychnia a pale blue color, and with respect to iodic acid Dr. R. Southey has found that when strychnia is heated with a saturated solution of iodic acid a rose-pink color is produced. (*St. Bartholomew's Hosp. Reports*, 1874, p. 301.) He proposes this as a test for strychnia even more delicate in reaction than the color test above described. Before accepting it as such, it will be necessary to prove that no other alkaloid or other substances produces a similar effect. A trace of anilin or morphia, of an alkaline sulphocyanide or saliva, will readily decompose iodic acid, and give a reddish tint from the separation of iodine. The fallacies to which the use of iodic acid may lead, have been well exposed by Dr. Dupré. (*Guy's Hosp. Reports*, 1863, p. 323.)

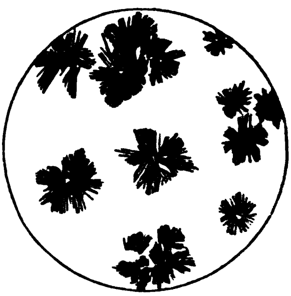
It has been rather dogmatically stated that the color-reactions, as above described, are peculiar to strychnia. For all practical purposes they may be so; but Dr. Fraser has shown that the African poison akazga produces similar colors, and like strychnia it also causes death by tetanic convulsions. (See p. 670.) The alkaloid of curara (*curarina*), according to the observations of Dr. Voisin and others, produces similar color-reactions with sulphuric acid and bichromate of potash; but as sulphuric acid alone gives a blue color to this alkaloid (*Ann. d'Hyg.*, 1866, vol. 2, p. 155), the fallacy arising from the color test is easily avoided.

FIG. 74.



Crystals of sulphocyanate of strychnia magnified 124 diameters.

FIG. 75.

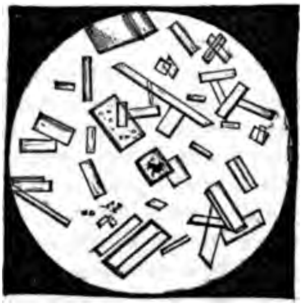


Crystals of chromate of strychnia, magnified 124 diameters.

The *salts* of strychnia are very soluble in water, and these solutions are precipitated by the chloriodide of potassium and mercury, the ioduretted iodide of potassium and tannic acid. They are not soluble in ether or chloroform; hence organic liquids containing these salts may be freed from fatty and coloring matters by first agitating them with these liquids. The salts yield crystalline precipitates with a large number of substances. The alkalies and alkaline carbonates, if diluted, separate the pure alkaloid in long slender prisms. (See Fig. 73.) Picric or carbazotic acid, recommended by Dr. Guy, is even a more delicate precipitant of a solution of strychnia. It gives small tufts or groups of stellated crystals. Chromate of potash produces similar tufts of chromate of strychnia. (Fig. 75.) According to Dr. James Gray, the sulphocyanate of strychnia in crystals may be produced in solutions containing not less than $\frac{1}{7000}$th part of strychnia. (Fig. 74.)

Dr. Filhol recommends as delicate precipitants of solutions of strychnia, chlorine, and chloride of gold, taking care that there is no alcohol in the liquid to be tested. Chloride of gold thus slowly precipitates in a crystalline form even the $\frac{1}{800}$th part of a grain of strychnia. A negative reaction with this test, would finally show that no detectable quantity of strychnia was present in solution. When these precipitates, drained of water, are treated with concentrated sulphuric acid, they are dissolved, and to this mixture a crystal of acid chromate of potash may be added to bring out the blue coloration peculiar to strychnia. In the case of chromate of strychnia the sulphuric acid produces at once the series of colors in contact, and thus this precipitant presents a great advantage over the others. Under a low power of the microscope a change of color in the smallest fragment of crystal may be distinctly seen.

FIG. 76.



Crystals of salicin, magnified 124 diameters.

Strychnia has been fatally mistaken for santonin (*Lancet*, 1870, vol. 1, p. 598), salicin, and jalapin, and has caused death on several occasions. Jalapin does not crystallize, but the well-defined crystalline forms of salicin are shown in the annexed illustration. (Fig. 76.) They are very different from those of strychnia. For the crystalline forms of santonin the reader is referred to Fig. 64, p. 653.

These two vegetable principles differ from strychnia in their properties. When heated in close tubes, they give off *acid* vapors. Salicin is soluble in water. Santonin is not soluble in water, but is dissolved by alcohol. Tannic acid and the chloriodide of potassium and mercury do not precipitate the solutions, while they readily precipitate those of strychnia. Nitric, iodic, and sulphomolybdic acids have no effect upon either. Sulphuric acid does not change santonin, but gives a rose-red color to salicin. Salicin undergoes no change with nitric and iodic acids, but acquires a deep maroon color from sulphomolybdic acid. The crystals of santonin closely resemble (in microscopical appearance) those of salicin, but they are distinguished from salicin and other alkaloids and principles by acquiring a brilliant yellow color on exposure to sunlight, without undergoing any change of form.

Organic Mixtures—The Tissues.—A similar method of research answers for both. If there is much water present, this must be evaporated to an extract in a water-bath. The strychnia may be then obtained by filtration in a state fit for preliminary testing.

An organic liquid containing strychnia, dissolved, will yield a precipitate after filtration by the chloriodide of potassium and mercury, and the ioduretted iodide. The liquid will also have an intensely bitter taste. The dialytic process by a tube (see *ante*, p. 154) applied to a small quantity of the liquid, will be found a convenient method of preliminary testing for the presence of strychnia. It will allow of the separation of strychnia, when in combination with an acid and in a state of solution, from blood, mucus, and other mixed organic matters present in the stomach or in articles of food. The tests above mentioned may be applied to the dialyzed, in place of the filtered liquid, and another small portion may be evaporated to dryness on opal glass or porcelain. The dry residue may be tested by sulphuric acid and oxide of manganese. If the results indicate by color the presence of strychnia, the whole of the liquid or the extract, dissolved in water, should be acidulated with acetic acid, and digested at a moderate temperature for some hours. It may be then transferred to the large dialyzer (*ante*, p. 155). In the course of six or eight hours the strychnia will be transferred as acetate into the dialyzed liquid. This is concentrated by evaporation to the smallest possible bulk, and while still acid is shaken with twice its volume of chloroform, which removes oily and other organic matters, but not the salt of strychnia. The acid liquid is next separated from the chloroform, by decantation or a pipette, and rendered slightly alkaline with ammonia. It is then again agitated with three or four times its bulk of chloroform. This dissolves the strychnia separated by ammonia, and on evaporating the chloroform spontaneously the alkaloid is obtained in a dry or crystalline state fitted for testing. This process has been most successfully carried out by Dr. St. Clair Gray, of Glasgow (*On Strychnia*, 1872, p. 75).

For separating the alkaloid from the *tissues*, Dr. Gray has found that digestion of the sliced tissue at 70°, for twenty-four hours, with acetic acid and water only, is sufficient for the purpose of dialysis.

Stas's Process.—This, more or less modified, has been generally employed by analysts for the separation of strychnia, especially as it exists in the tissues. The principle of its operation consists in dissolving the strychnia by a gentle heat out of the tissue or organ, previously pulped or finely sliced, by means of rectified spirit mixed with a small quantity of acetic acid. The liquid is strained, and the residue well pressed and washed with alcohol; the acid solution of strychnia thus obtained is concentrated in a water-bath at a low temperature. The concentrated liquid is filtered and neutralized by ammonia, a slight excess of alkali being added. The alkalized liquid is then shaken in a long stoppered tube, with three or four times its volume of ether or chloroform, or a mixture consisting of three parts of ether and one of chloroform. These liquids dissolve the strychnia set free by the alkali. The ethereal solution is separated from the watery liquid by a pipette or by a stoppered tube, and submitted to spontaneous evaporation, when, if strychnia is present, the alkoloid will be obtained, but generally associated with oily and other organic matters, which interfere with the production of crystals. The impure residue left by the ether is heated in a water-bath, with a few drops of strong sulphuric acid; this destroys the organic matter without materially affecting the strychnia. Water is added, and the acid liquid is filtered through paper, neutralized by ammonia, and again treated with ether, when strychnia will be procured in small and slender colorless prisms. The crystals, after an examination by the microscope (see Fig. 73, p. 685), are treated with sulphuric acid and peroxide of manganese, and the color reactions of strychnia, if the alkaloid is present, will then appear. By this method I have detected strychnia in the liver of a person who died from this poison, although the organ was in a highly putrescent state.

The Chromate Process.—It is many years since Mr. Horsley, of Cheltenham, first recommended the chromate of potash as a precipitant of strychnia and the subsequent production of the color-reactions by the addition of sulphuric acid to the dried precipitate of the chromate of strychnia. This process by precipitation is deserving of more attention than it has hitherto received from toxicologists. Dr. F. Mohr found that one grain of pure strychnia dissolved by heat in diluted sulphuric acid, precipitated by bichromate of potash, and the precipitate washed until the water was colorless and free from bitterness, yielded 1.28 grain of acid chromate of strychnia; three grains of strychnia gave 3.811 of acid chromate of strychnia, containing 78.77 per cent. of strychnia. He found that a solution containing only $\frac{1}{1000}$th part of strychnia gave, after standing for a night, a perceptible precipitate with the acid chromate. He observes that this method is singularly free from objection. The acid chromate of potash produces similarly colored precipitates with the salts of lead and baryta. These bodies are, however, excluded by the processes through which strychnia is extracted. (*Chemische Toxikologie*, von Dr. F. Mohr, 1874, p. 131.)

So insoluble is the chromate of strychnia that I have found a solution of one grain of strychnia in six ounces of water precipitated by it. The strychnia precipitate is increased in bulk; it is entirely freed from organic matter.

By weighing the dry precipitate, the weight of strychnia present may be determined. Care must be taken before adding the chromate that neither alcohol, ether, or chloroform is present, and that the liquid has an acid reaction.

The chromate of strychnia thus obtained, contains the proportions of strychnia and chromic acid well fitted to give with sulphuric acid the color-reactions in the greatest purity and intensity. I have found that brucia and a solution of curara are equally precipitated by the acid chromate of potash. This, however, will not affect the general application of the process. The acid liquid obtained by the methods above mentioned supposed to contain strychnia, is simply concentrated to the smallest bulk and treated with the acid chromate. A precipitate may be at once formed, or it may require some hours for its deposit, according to the proportion of strychnia present. It may be crystalline or amorphous. When deposited, it may be collected, washed, and dried. If small, the drying should take place on opal glass or a porcelain capsule. When dry, the addition of sulphuric acid will bring out at once the color-reactions of strychnia. The application of the acid to the dry filter will also show the colors.

Whatever process may be adopted for the extraction of strychnia from organic liquids, the analyst should bear in mind that it is better to employ the tests on a small quantity of strychnia in a pure state, than upon a large quantity in an impure state.

The reader will find a full account of the processes for strychnia in organic solids and liquids in some valuable papers published by Dr. Wormley in the *Ohio Medical Journal*, January, 1864, p. 19, and March, 1864, p. 119, since republished in his *Micro-Chemistry of Poisons*, New York, 1867, p. 534. This gentleman describes a case in which, from a misapplication of the process, strychnia was sworn to be present, when, from the chemical method pursued, the alkaloid could not possibly have been separated ; and two instances have fallen within my own knowledge, where the colors produced by sulphuric acid and bichromate of potash on the concentrated and dry organic contents of the stomach, were referred to strychnia, when they were really due to the decomposition of the bichromate employed in contact with organic matter! The detection of this poison in the stomach or the tissues will depend on the same conditions as those observed in other cases of poisoning. If a person takes a large dose and dies quickly, a residuary portion may be readily found. In *Reg.* v. *Burke* (Clonmel Summer Assizes, 1862), the prisoner administered strychnia to his wife in Epsom salts. She died in about half an hour under the usual symptoms. Dr. Blyth extracted more than three grains of poison from the contents of the stomach. If a small dose has been taken, and the person has survived some hours, it is probable that none will remain in the stomach. A decomposed state of the viscera and their contents does not appear to interfere with the detection of this poison. It has been suggested that the presence of morphia counteracts the color tests ; but unless in admixture in large proportion with the strychnia, it does not interfere with the production of the changes of color. Besides, in the process of separation described, morphia is not readily taken up by ether or chloroform. The presence of morphia in strychnia is readily detected by iodic acid and sulphomolybdic acid (page 555).

The principal salts of strychnia are the *Hydrochlorate*, *Acetate*, and *Sulphate*. All are eminently poisonous, and owing to their great solubility in aqueous liquids, they are rapidly absorbed. The respective acids may be recognized by the usual tests.

The *liquor strychniæ* is the only preparation of this alkaloid which finds a place in the British Pharmacopœia. It consists of strychnia dissolved as hydrochlorate in rectified spirit and water in the proportion of four grains to one ounce—one grain to two drachms ($\frac{1}{120}$th). The dose for an adult is from five to ten minims.

Persons may die from strychnia, and no trace of the poison be found in the body. In a case of poisoning by this alkaloid, which was the subject of a trial for murder at Perry Co., Pa., in the April term of

1861, Dr. Reese, of Philadelphia, made separate analyses of the contents of the stomach and the contents of the intestines, as well as of the tissues, and each one of these was repeated to avoid a possible error. Yet there was no evidence of the presence of strychnia either by the bitter taste of the final extract, or by the color tests. The witness, by a comparative experiment, satisfied himself that he could detect the half-millionth of a grain (*Amer. Jour. Med. Sci.*, Oct. 1861); but in this power of detecting so small a quantity of strychnia in a pure state, he had already been anticipated by Mr. W. Copney. (*Pharm. Jour.*, July, 1856, p. 24.) In Dr. Reese's case, the quantity taken by deceased was unknown; the woman lived five or six hours, and the body was not examined until six weeks after death. A small but fatal dose, and the duration of the case, will sufficiently account for the negative results without resorting to any other hypothesis. In the case of a Mrs. Salter, who died from a dose of strychnia in September, 1869, death probably took place within two or three hours; but the most careful examination made of the stomach and liver by Mr. Horsley, of Cheltenham, led to a negative result. Strychnia, in the opinion of all the medical witnesses, was the cause of death, but no trace of strychnia could be detected in the body by one well qualified to detect it. There was some reason to think that the poison had been taken in solution, but, even under these circumstances, it must have been rapidly absorbed, diffused, and eliminated.

When death has been caused by small doses applied under the skin, it is not likely that the poison will be found in the tissues. The following experiment was performed in May, 1864. One-eighth of a grain of acetate of strychnia in coarse powder was placed in the cellular membrane of the neck of a rabbit. In nine minutes the animal was seized with a sudden convulsion and fell on its side; its fore and hind legs were rigidly stretched out, and its body passed into a state of opisthotonos. It had a succession of fits, and died in one of them, in twenty minutes after the commencement of the symptoms. One-half of the powdered acetate was still found in the wound, showing that the rabbit had been killed by the sixteenth part of a grain. Of course the *residuary* strychnia was easily detected in the wound; it was plainly visible to the eye. On applying the usual process for detecting strychnia to the heart, blood, and the liver, the tests failed to indicate the slightest trace of the alkaloid. The ethereal liquid left no crystalline residue of any kind. This result does not show that strychnia is not absorbed, but it proves that under certain conditions it cannot be detected in the organs of the body, in cases in which, beyond all doubt, it has destroyed life. In instances in which death has been caused by nux vomica, which contains only one per cent. of the poisonous alkaloid, so far as I know, strychnia has not been found deposited in the tissues.

The following case, which occurred at Liverpool in April, 1864, of which the particulars were communicated to me by Dr. J. B. Edwards, shows the rapidity with which the poison may be diffused and deposited in the tissues, when a large dose has been taken. A strong healthy man, æt. 43, placed upon his dry tongue, at 10 P.M., a powder which contained six grains of Dover's powder and *five grains* of strychnia (dispensed by mistake for five grains of James's powder). He complained of a bitter taste, asked for an orange, and he found, on sucking this, that it increased the bitterness—the acid juices of the orange instantly dissolved the strychnia, and thus favored its early absorption. In *fifteen minutes* he went to bed; twitchings of the muscles then came on, and the patient, from previous experience in taking strychnia as a medicine, was fully aware of the cause of the symptoms. He complained of his bowels being drawn up; he drew his knees up as if to his mouth, gave a yell, seized hold of a friend who was standing by, and became apparently unconscious (exhausted) for about five minutes. He then revived, but in a few minutes was again seized with a violent convulsion of a tetanic character, and he died in two or three minutes afterwards, a little over *half an hour* after taking the powder. Owing to a spasmodic closure of the jaws, he was able to speak only for a few minutes at a time; he was rational, but seemed to be in great terror. An inspection was made thirty-six hours after death, when the body was found to be unusually warm. The back and lower parts were much discolored, but the discoloration was easily removed by pressure, indicating a fluid state of the blood. The lower jaw was slightly drawn up; the body straight, not arched; the fingers slightly flexed, not clenched; the thumbs were fixed, but not bent into the palms of the hands. On opening the head, the scalp was found very much gorged with fluid blood. The membranes of the brain were also much congested, as well as the bloodvessels of the brain generally. There was no clot or coagulum, but a quantity of serous fluid had escaped from the surface. There was no softening of the substance of the brain or spinal cord. The lungs were healthy, but dark-colored from engorgement with blood; the heart was empty; its structure was soft; the liver was healthy; the spleen was gorged with fluid blood; the kidneys were congested. The other organs presented no appearance calling for notice. By chemical analysis, Dr. Edwards found strychnia in the stomach, the quantity being estimated at about one grain. He also found the poison in the tongue and the liver. He sent to me a portion of the liver, one kidney, and six ounces of blood. They were in a putrescent state, and, when examined about three months after death, eight ounces of liver yielded, by the process above described, prismatic crystals of strychnia, producing the usual color-reactions with sulphuric acid and peroxide of manganese, as well as with bichromate of potash. The quantity of strychnia thus obtained was small. The kidney and the blood did not give results on which any reliance could be placed.

This case shows that a large dose of strychnia rendered soluble will destroy life in half an hour—that within this short time four-fifths may be removed from the stomach, or at least not be discoverable there by careful chemical analysis after death—that in half an hour the poison may be distributed through the body and deposited in the soft organs, although no satisfactory evidence of its presence could be obtained from less than half a pound of animal matter. The strychnia found in the tongue was probably a portion of the powder swallowed, which still remained there. It may be further remarked that, although the deceased took in the James's powder $\frac{6}{10}$ths of a grain of opium, no morphia was present in the crystalline residue obtained from the liver. (For numerous additional facts and cases connected with this question, see *Guy's Hospital Reports*, Oct. 1856, p. 326.)

In the case examined by Casper (pp. 677, 679) the deceased admitted that he had taken between five and six grains of strychnia. He lived three hours and a half, and on analysis Dr. Sonnenschein procured from the stomach three grains of the poison. He did not, however, succeed in extracting any from the blood or tissues. In the cases of infants or children destroyed by small doses, some may be found in the stomach, and too little deposited in the tissues to allow of separation. In *Reg.* v. *Bailey and Barry* (Gloucester Assizes, Dec. 1873), the prisoners were charged with the murder of an infant by strychnia given in a powder. The child died rapidly, and the symptoms were such as strychnia would produce. No strychnia could be detected in the body, and its absence was explained by the witness to be partly due to the smallness of the fatal dose, and partly to the removal of the poison by absorption. The prisoners were convicted and executed, the chemical evidence being supplemented by the physiological.

If strychnia has been administered in *pills* and, after death, the stomach has been cut open and the contents lost, there will be little hope of discovering the poison, although in *Reg.* v. *Palmer* (C. C. C., May, 1856) some experts affirmed that this state of things would not materially interfere with the chemical process for its detection! The diffusion of the contents of the stomach through the whole of the small and large intestines and their feculent fluids, was on this occasion treated as a comparatively unimportant matter, creating only some delay in obtaining the results! It need hardly be observed that these are speculative opinions, and that the experts who uttered them had never met with or seen a case by which the truth of these statements could be fairly tested. In those inspections in which there has been such criminal interference or culpable neglect, as in that of J. P. Cook, the only course for an analyst is to seek for the poison in the tissues. This case, concerning which so much has been said and written in utter ignorance of the scientific facts, settled nothing in reference to the presence or absence of strychnia in the body of a person poisoned by this substance. Except the actual destruction of the stomach itself, everything had been done which possibly could be done in order to render a chemical analysis utterly fruitless. It cannot therefore be taken as a fair precedent in any sense for the results of a proper medico-legal research in poisoning with strychnia. The greatest reproach to Dr. Rees and myself would have been that we should have detected strychnia under circumstances in which any honest and unprejudiced analyst would have pronounced its detection next to impossible.

It is satisfactory to find that a correct view of this remarkable case, and its true bearings on science, has been shown by continental jurists. In an analysis of this case, by the late Professor Casper (Horn's *Vierteljahrsschrift*, Juli, 1864, p. 26), not only are the chemical results justly regarded as negative, by reason of the gross mismanagement of those who inspected the body, but the post-mortem appearances themselves, for a similar reason, are considered as throwing no light upon the effects of strychnia on the body. The active co-operation of a professional poisoner, at the examination of the body of his victim, is an exceptional circumstance, even for the liberal customs of this country! It was only in the natural course of things that attempts were actually made to defeat a chemical analysis, but when such attempts have proved successful, it is not usual to find scientific witnesses actively struggling for the honor of defending a prisoner, not because the deceased did not die from poison, but because it was not chemically detected in his body. The failure of this branch of evidence furnished a favorable opportunity for introducing a variety of ingenious speculations on the cause of death. One of the medical witnesses for Palmer could see in the whole case nothing but angina pectoris, another only epilepsy with "tetanic complications." One expert assigned death to brucia and another to morphia! These conflicting and dissimilar views would justify the public in placing no confidence in medical opinions in cases of this kind. Assuming that there had not been a criminal interference with the dead body on the part of the prisoner Palmer, the position assumed, that strychnia, if the cause of death, must always, and under all circumstances, be found in the dead body, is simply untrue. Its detection in the body, properly verified, is a proof that it has been taken; the symptoms in their commencement, progress, and termination will furnish irrefragable proof that it has acted as a poison; but its non-detection does not prove that it has not destroyed life.

The case of J. P. Cook, here referred to, is marked by this peculiarity. It was watched from the beginning to the end by a medical friend of the deceased. The witness Jones saw the pills given to the deceased by the prisoner, but he had no suspicion at the time that they contained strychnia. The evidence respecting the symptoms which he observed was as follows: Three-quarters of an hour after taking the pills deceased appeared comfortable, but in ten minutes more (fifty-five minutes after taking them) he (the witness) was roused by deceased, who was sitting up in bed, saying he was going to be ill. He asked his friend to rub his neck, and to send for his medical attendant Palmer. After swallowing two other pills (said to contain ammonia), which the prisoner had brought with him, deceased fell back on the bed in convulsions. He said he should be suffocated. They tried to raise him; but he was so stiffened out with spasms that it was impossible. He then said, "Turn me over." He was turned on his side, and he died in a few minutes. The medical man described the symptoms as those of *tetanus;* every muscle of the body was simultaneously stiffened. When his neck was rubbed, the muscles of the head and neck were found to be affected with violent spasms; his head was thrown back; his hands were clenched; and his arms were in a state of rigidity. His jaw was closed and fixed. His body was stretched out and rested on the head and heels in a state of opisthotonos. The symptoms, therefore, in this second and fatal attack, came on in about an hour after deceased had taken the suspected pills; and he died in from sixteen to twenty minutes after their commencement.

The body of the deceased was inspected on the 26th November: six days after death; it was then in a state of rigid spasm. The viscera were stated to be universally in a sound and healthy condition. The membranes of the brain were a little congested; the heart was empty, and the blood generally dark and fluid. The mucous membrane of the stomach, as well as that of the intestines, was partially congested. There was no appearance of any disease to account for death.

Comparing these symptoms with those which have been met with in strychnia-poisoning, it is impossible to come to any other conclusion than that strychnia was the cause. This was the view taken at the trial by Sir R. Christison, the late Sir B. Brodie, and other medical men of experience. One medical expert admitted that it was a case of poisoning, but assigned morphia as the poison! This, however, would not account for the violent tetanic paroxysms and rapid death. Others, in accordance with a statement made by the prisoner before his execution, assigned the symptoms and death to *brucia*. But there is no proof that the prisoner had had any brucia in his possession, and it would have required six times as much brucia as of strychnia to produce the fatal symptoms. On the other hand there was distinct evidence that the prisoner had secretly procured, from a medical friend, three grains of strychnia shortly before the first attack of the deceased on the Monday night, and that he had secretly purchased six grains of this poison on the morning of Tuesday, the day on which the deceased was taken with the second and fatal attack. No reasonable motive could be suggested by the astute counsel who defended the prisoner, the late Mr. Justice Shee and Mr. Justice Grove, for his secretly procuring nine grains of this deadly poison from two different sources within twenty-four hours of the death of the deceased, nor could any explanation be given of what had become of it! The prisoner had, therefore, the motive, the means, and the opportunity, of perpetrating the crime, while death by suicide was wholly inconsistent with the facts.

For a more complete history of the *medical* facts in the memorable case of Cook I must refer the reader to a paper on Poisoning by Strychnia, *Guy's Hospital Reports*, October, 1856; *Pharm. Journal*, July, 1856, p. 6 (from the pen of the late Jacob Bell). The most able legal analysis of it by any English writer which I have seen has been published by Mr. J. F. Stephens, in his *General View of the Criminal Law of England*, 1863, p. 357. Of the foreign reports, one by M. Tardieu, in the *Ann. d'Hygiène* for 1856, vol. 2, p. 371, and 1857, vol. 1, p. 132, and the other by Professor Casper, in Horn's *Vierteljahrsschrift für ger. Médicin*, etc., 1864, vol. 2, p. 1, are the most correct in their medical and medico-legal details.

Strychnia in Organic Solids.—From the vermin-killers of Butler and Battle, the strychnia may generally be readily separated by means of alcohol, and procured in a crystalline form for the application of tests. If the vermin-killer is covered with Prussian blue one or two drops of sulphuric acid will remove the color, and the oxide of manganese may be then added. The color-reactions are then as well marked as with pure strychnia.

In cases in which the poison is contained in pills or powders, having much organic matter soluble in alcohol, it will be advisable to employ dilute sulphuric acid for its extraction. It is a remarkable fact that strychnia itself is not acted on in the same degree by sulphuric acid as organic matter, or even as other poisonous alkaloids.

CEREBROSPINAL POISONS.

CHAPTER LXXI.

COMMON HEMLOCK—SYMPTOMS AND APPEARANCES—CONIA—WATER-HEMLOCK—ŒNANTHE CROCATA—ÆTHUSA CYNAPIUM.

THE poisons belonging to this class are considered to affect both the brain and spinal marrow directly, and the heart and other organs indirectly. They are derived chiefly from the vegetable kingdom, and owe their noxious properties to the presence of an *alkaloid*, which admits of separation. The effects of the alkaloid are not only more intense than those of the vegetable from which it is extracted, but sometimes different in kind. Thus the vegetable may produce irritation, and inflammation of the stomach and bowels, before the cerebrospinal symptoms are manifested, while the alkaloid may operate on the brain and spinal marrow immediately after absorption. Among the alkaloids, cónia and aconitina are the most powerful. In some cases they appear to act as spinal poisons, for they have caused paralysis or convulsions, while the intellect has remained unaffected. Conia operates in two opposite modes on the spinal marrow,—it produces a state analogous to tetanus, or more commonly it causes a complete paralysis of the muscular system without materially affecting sensibility. It also acts locally as an irritant. The term narcotico-irritant, however, is not appropriate to this class of poisons, for many of them operate without causing stupor or insensibility; and, so far as the alkaloids are concerned, there is no marked degree of irritation of the stomach or bowels.

The symptoms caused by the leaves or roots of the vegetable poisons are generally manifested within an hour; those caused by the vegetable alkaloid, within a few seconds or minutes. As a summary, the symptoms from this class of poisons may be stated to be giddiness, delirium, coma, paralysis of motion, or sensation, or both, with convulsions of a clonic or tetanic kind. The pupils of the eyes are commonly dilated. Occasionally there is vomiting, and severe pain in the stomach and bowels. The poisons in the state of leaves or root have in general a hot, bitter, numbing, or other well-marked taste, so that they cannot be criminally administered without exciting suspicion. Murder by monkshood has, however, been perpetrated by the substitution of the leaves of this plant for other vegetables at a meal, and in one instance in which I was consulted a child was destroyed by sipping a decoction of hemlock. Fatal accidents have occurred by reason of persons eating the roots of the œnanthe crocata, the taste of which they found palatable, the plant having been mistaken for wild celery or wild parsnip.

As there is a great variety in the effects produced by this class of poisons, so the appearances in the bodies of those who have been killed by them have been subject to variation. In some instances the stomach and intestines are congested or inflamed; in others not. When the person has died under symptoms of narcotism, traces of cerebral congestion are occasionally found, and when the symptoms have been those of asphyxia from paralysis of the muscles of the chest, there has been congestion of the heart and lungs. Microscopically the blood is not altered, but it is generally dark and liquid.

Analysis.—Most of the cerebrospinal or narcotico-irritant poisons owe their deleterious effects to the presence of an alkaloidal principle similar to morphia, and susceptible of isolation by complex chemical processes. There is, however, considerable difficulty in extracting these alkaloids from the respective vegetables; and, when extracted, the chemical differences among them, in respect to the action of tests, are very slight. Indeed, better evidence of the poisonous nature of a suspected liquid or extract has been hitherto derived from the administration of a portion of it to animals, than from the application of chemical tests. In a medico-legal point of view, there are, with few exceptions, no chemical tests for these vegetable alkaloidal poisons in organic liquids or solids, upon which reliance can be placed. Some pretenders to minute accuracy of analysis have applied their tests to the isolated alkaloid, and have led the public to believe that it was just as easily tested in the absorbed state in the blood and soft organs of the dead body as any of the more common poisons. Experience, however, shows this statement to be erroneous. When the vegetable has been used, either in the shape of seeds, leaves, berries, or root, then valuable evidence may be sometimes procured by searching with or without the aid of a good microscope for the botanical characters of the plant; these parts of the plant, from their indigestible nature, may be found in the vomited matters or evacuations during life, or in the stomach and bowels after death. The broken leaves may be separated by washing, as they are quite insoluble in water; they may be therefore easily collected, dried on mica and examined by the microscope, which, under the hands of a skilful botanist, may thus reveal the nature of the poison. This source of evidence will, however, often fail, owing to the poison having been taken in the form of extract, infusion or decoction.

Treatment.—The treatment of a case of cerebrospinal poisoning consists in promoting early vomiting by emetics, or in drawing off the contents of the stomach when this is possible by the stomach-pump. If there should be reason to suppose, from the seat of pain, that the poison has descended into the bowels, then laxative injections or castor oil may be used. Recoveries have taken place when the poison has been thus removed, even although symptoms had set in. Cold affusion, or stimulants, may occasionally be required; the patient, if inclined to sleep, should always be kept roused. There is no certain chemical antidote to any of these poisons. Tannic acid precipitates all the alkaloids; hence it has been strongly recommended as an antidote. No injury can follow its exhibition; and a decoction of black tea will be a good substitute for oak-bark or galls. With respect to electricity, Ducros found that the negative current was beneficial to animals poisoned with strychnia or brucia; while the positive current produced convulsions, and accelerated death. (Canstatt, *Jahresbericht*, 1844, vol. 5, p. 297.) M. Bouchardat advises the antidotal use of a solution of iodine in iodide of potassium, the proportions being three grains of iodine, and six grains of iodide of potassium, to sixteen ounces of water. Half a wineglassful of this solution should be administered occasionally, and vomiting promoted in order to eject the compound formed. This antidote is supposed to operate by forming an insoluble compound with the alkaloid, except in the case of digitalin. (Bouchardat, *Annuaire de Thérapeutique*, 1847, p. 301.) Dr. Garrod recommends, as an efficacious remedy in the early stage of this form of poisoning, the free use of animal charcoal. (*Med. Times and Gaz.*, Dec. 5, 1857, p. 590.) There is no doubt that animal charcoal has a tendency to remove alkaloids from liquids. It has thus been found to precipitate strychnia, and it is equally efficacious with atropia and aconitina. When, however, the poison is already in the blood, this and other suggested antidotes would be of no avail. If the poison has been taken in the form of slices of root, as in the case of aconite, it is not easy to perceive how any antidote in the stomach could arrest or prevent the effects of the poison in the blood. In a case in which I was consulted, the slices of the roots of aconite were found unchanged in the stomach after death; the poison had been imbibed from their surface by the absorbents of the mucous membrane of the stomach. Animal charcoal, however, is so far a safe remedy: it can do no injury, and it may act beneficially by partially precipitating the alkaloid if dissolved, or by enveloping it and sheathing the coats of the stomach from its operation, if contained in the roots, leaves, or seeds of the plant. The removal of the poison either by emetics and purgatives, or by the stomach-pump, must, however, in all cases be the primary object of treatment.

COMMON OR SPOTTED HEMLOCK (CONIUM MACULATUM).

This is a well-known hedge plant, which grows abundantly in most parts of Great Britain. Its effects on man and animals prove that it possesses active poisonous properties, which reside in the seeds, leaves, and roots, and may be extracted by water. Its energy varies, probably according to season and locality. The effects produced by hemlock have not been uniform. In some instances there have been stupor, coma, and slight convulsions, while in other cases the action of the poison has been chiefly manifested on the spinal marrow,—*i. e.*, it has produced paralysis of the muscular system.

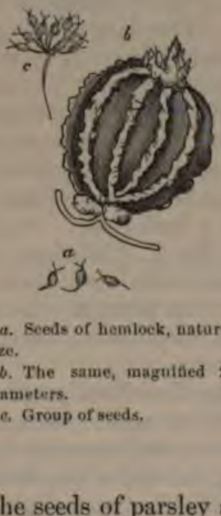
Symptoms and Appearances.—In a series of cases quoted by Orfila, several soldiers partook of hemlock in soup. Soon afterwards they all appeared to be intoxicated. One, who had partaken of the soup rather freely, became in less than two hours senseless; he breathed with difficulty; his pulse was hard, small, and slow; surface cold; and his face livid, like that of a person who had undergone strangulation. Emetics were administered, with temporary relief, but he became again unconscious, lost the power of speaking, and died in three hours after partaking of the soup. On inspection, the stomach was found half filled with a quantity of pulpy matter, and there were some red spots on the mucous membrane, near the intestinal end. The vessels of the brain were gorged with blood, which was quite liquid. (*Op. cit.*, 4ème edition, vol. 2, p. 427.) In this case the operation of the poison was chiefly manifested on the brain. Dr. J. H. Bennett met with a case which illustrates the other mode of action. A man ate a large quantity of hemlock-plant by mistake for parsley. In from fifteen to twenty minutes there was loss of power in the lower extremities; but he apparently suffered no pain. In walking, he staggered as if he was drunk; at length his limbs refused to support him, and he fell. On being raised, his legs dragged after him, and, when his arms were lifted, they fell like inert masses, and remained immovable. There was perfect paralysis of the upper and lower limbs within two hours after he had taken the poison. There was loss of power of swallowing, and a partial paralysis of sensation, but there were no convulsions, only slight occasional motions of the left leg; the pupils were fixed. Three hours after eating the hemlock, the respiratory movements had ceased. Death took place in three hours and a quarter; it was evidently caused by gradual asphyxia from paralysis of the muscles of respiration, but the intellect was perfectly clear until shortly before death. On inspection, there was slight serous effusion beneath the arachnoid membrane. The substance of the brain was soft; on section there were numerous bloody points, but the organ was otherwise healthy. The lungs were gorged with dark fluid blood; the heart was soft and flabby. The stomach contained a green-colored pulpy mass resembling parsley. The mucous coat was much congested, especially at its greater end. Here there were numerous extravasations of dark blood below the membrane, over a space of about the size of the hand. The intestines were healthy, here and there presenting patches of congestion in the mucous coat. The blood throughout the body was fluid and of a dark color. A portion of the green vegetable pulp was identified by Sir R. Christison as part of the leaves of the *Conium maculatum.* Some of the leaves bruised in a mortar, with a solution of potash, gave out the peculiar odor of the alkaloidal principle Conia. (*Ed. Med. and Surg. Jour.*, July, 1845, p. 169.)

Dr. Skinner had an opportunity of observing the effects produced by hemlock on five children, varying from five to eight years of age. (*Liverpool Med.-Chir. Jour.*, July, 1858.) The symptoms were dryness, with a feeling of constriction in the throat, headache, disposition to sleep, pupils dilated, pulse small and weak, the impulse of the heart weak, breathing slackened, a general paralysis of all the voluntary, and ultimately of all the involuntary muscles, the power of swallowing being the first, and of breathing the last, to fail. At the time of the general paralysis coming on, there was more or less coma with foaming at the mouth, and death appeared to be the result of coma. In two of the cases, the brain was the first organ affected, and the paralysis of the legs never appeared, as the children ran home, complained of sleepiness and desired to be put to bed.

In a case which occurred to myself, which was a subject of a trial for murder (*Reg.* v. *Bowyer*, Ipswich Summer Assizes, 1848), the child died in one hour, after swallowing part of a teacupful of a decoction of hemlock, alleged to have been administered by the mother. The child sipped the decoction until it lost the power of holding the cup; it became insensible and paralyzed, and died in the chair in a sitting posture. There were no morbid appearances, and no hemlock leaves were found in the body, these having subsided in the cup, and being left in the dregs. The child had been poisoned by the upper stratum of clear liquid, and the residue had been thrown away. The mother was acquitted for want of proof of criminality, the death of the child having taken place in secrecy.

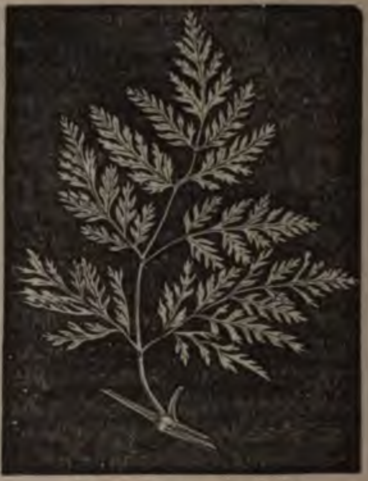
Analysis.—Hemlock is known from most other plants which resemble it by its large, round, smooth stem, with dark, purple spots. The leaves are of a dark-green color, and smooth and shining. Every portion of the plant has a peculiar and disagreeable smell when bruised, resembling cat's urine; an odor is brought out when the stem, leaves, or seeds, are rubbed with a solution of caustic potash, which has been compared by some to the odor of mice. The *seeds* of hemlock (Fig. 77) are peculiar in their form, and are easily distinguished from the seeds of parsley and of other umbelliferous plants. A person may be poisoned by a decoction of *leaves* of hemlock, and no leaves be found in the stomach or bowels (case of Bowyer, *supra*). In this case the stomach had been emptied and the contents lost before it was sent to me! No trace of conia was found. The prisoner first gathered the

FIG. 77.



a. Seeds of hemlock, natural size.
b. The same, magnified 20 diameters.
c. Group of seeds.

FIG. 78.

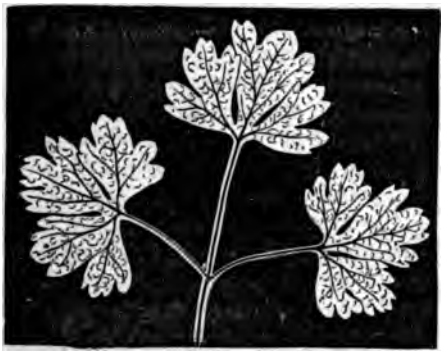


Leaves of common hemlock.

Anthriscus sylvestris by mistake for *Conium maculatum*, but it was proved that she had afterwards gathered the leaves of hemlock. A leaf of each of these plants was copied by photography, and produced as evidence in court.

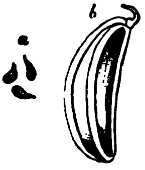
As the determination of the presence of fragments of *leaves* in poisoned liquids, or in the contents of a stomach, may be of importance as evidence, we subjoin an illustration of hemlock leaves, engraved from a photograph of the living plant (Fig. 78). The appearance and smell of the leaves, either when bruised or when rubbed with a solution of potash, will greatly aid a medical witness in forming a judgment, as there are many umbelliferæ which bear a close resemblance to hemlock in the form of their leaves. Among these, however, it is impossible to rank common parsley (Fig. 79). It is hardly credible that a mistake of this kind should be made, yet through carelessness and ignorance accidents have occurred. In August, 1864, a lady and two of her children, residing in the neighborhood of Liverpool, were seized with symptoms of poisoning soon after dinner. The medical gentlemen who were called in examined the remains of some soup which had been eaten for dinner, and they detected fragments of the leaves of hemlock amongst the herbs which had been used, by mistake for parsley, to flavor the soup. Under treatment, the symptoms abated in a few hours, but these persons did not entirely recover until after two or three days. It turned out that the hemlock had been gathered in a garden belonging to the family, where it was growing side by side with parsley. As the parsley was raised from seed, it is probable that hemlock seed had been accidentally mixed with it by the seedsman, and thus the accident had occurred. We subjoin an illustration from a photograph of a leaf of parsley; also an illustration of the seeds, by which the differences between hemlock and parsley will be at once apparent (Figs. 77 and 80).

FIG. 79.



Leaves of garden parsley, from a photograph.

FIG. 80.



Seeds of garden parsley.
a. Natural size.
b. Magnified.

CONIA.

The alkaloid of hemlock is known under the names of conia, conein, conicin, and conicina. It is a volatile liquid like nicotina. It has a pale yellow color, but is slowly darkened by exposure to air. It consists, like prussic acid and nicotina, of carbon, hydrogen, and nitrogen, without any oxygen. Its specific gravity is 0.89. It has the powerful pungent odor of hemlock, with a bitter and intensely acrid taste; it is irritating to the skin. It is transparent, oily-looking, and floats on water, in which it is not very soluble. It combines with diluted acids to form poisonous salts. It exists in all parts of the plant, but an alcoholic extract of the seeds yields the largest quantity. The proportion of conia in the plant probably varies at different seasons of the year, a fact which will account for the root having been occasionally eaten with impunity.

The common *extract* of hemlock, which owes its properties to this alkaloid, is liable to vary much in strength, according to the mode in which it has been prepared; when overheated, there is a great loss of conia. The presence of this alkaloid in the extract may be readily determined by triturating it with caustic potash; if present it is immediately set free, and may be recognized by its peculiar odor, resembling the smell of mice.

Sir R. Christison's experiments prove that conia, whether free or combined, is a potent poison. It produces, according to him, general palsy without insensibility, and with slight occasional twitches only of the limbs of the animal. (*Op. cit.*, p. 855.) The heart was not affected by the poison, as this organ pulsated even after other signs of life had ceased. Death appeared to be due to asphyxia, from the general paralysis of the respiratory muscles. A single drop of conia, applied to the eye of a rabbit, killed it in nine minutes; and three drops killed a strong cat in a minute and a half. The more recent observations of M. Verigo confirms Sir R. Christison's views, and show that conia does not act on the brain, or only in a slight degree. Its chief action is in the motor fibres of the spinal cord. In mammals large doses produce convulsions, while small doses produce paralysis of the extremities. It has no action on the heart or pulse, and there is no apparent change caused by it in the blood-corpuscles. (*Lancet*, 1871, vol. 1, p. 834.)

The effects of this alkaloid have been more recently observed by Dr. Van Praag. (Reil's *Journal für Toxicologie*, 1 H., 1856, p. 1.) He found it to accelerate respiration, to produce tetanic spasms, and an incurvated state of the limbs and feet, with emprosthotonos; before the cramps set in, there was a tremulous motion of all parts of the body. The symptoms of general paralysis preceding the spasms were manifested by the unsteady gait, the difficulty of standing, the drooping of the head, and the falling together of the knees. The senses and sensibility of the skin were not materially affected. The pupils were dilated and insensible. In some instances the brain was affected, but only slightly, the stupor or tendency to sleep was but slight, and the animal was easily roused. Vomiting or a disposition to vomit manifested itself in a few instances. On inspection, the brain and its membranes were found congested, the heart sometimes full, at other times its right cavities were empty. The blood was dark and liquid. The experiments of Orfila show that an animal, to which this alkaloid had been given, suddenly became powerless and fell; there were very slight convulsive motions in the limbs, without opisthotonos, lasting for about a minute; the animal then appeared to be in a collapsed state, and died in five minutes. (*Mémoire sur la Conicine,* 1851, p. 84, and *Ann. d'Hyg.*, 1851, vol. 46.)

Analysis.—*Conia* resembles nicotina and ammonia in its liquidity, volatile reaction, and in some of its chemical properties. It is a liquid of oily consistency, of a pale yellow color, powerfully alkaline, and has, when its vapor is diluted, a smell resembling that of mice, and an acrid bitter taste. It gives a volatile greasy stain to paper, and burns with a yellow flame and thick smoke. 1. It is not colored or affected by nitric, sulphuric, or hydrochloric acid; the last-mentioned acid produces with it dense white fumes of hydrochlorate of conia, and on heating the mixture this salt remains in prismatic crystals. 2. It is not readily dissolved by water, but floats on it in oily globules. 3. It is soluble in alcohol and ether, and this last-mentioned liquid removes it from its aqueous solution. 4. It gives a white precipitate with corrosive sublimate, and a yellow precipitate with arseno-nitrate of silver. 5. It precipitates brown oxide of silver from the nitrate; this is not dissolved by an excess, but the oxide is blackened and reduced. 6. Iodine-water gives a reddish-brown precipitate, which is redissolved; an excess of iodine-water causes a yellowish precipitate. 7. It gives a yellow crystalline precipitate with chloride of gold, but no precipitate with chloride of platinum. 8. Tannic acid precipitates it of a dingy white. 9. Gallic acid gives no precipitate, but slowly acquires a yellowish color. Its odor and insolubility in water, as well as several of the characters above mentioned, serve to distinguish it from nicotina and ammonia, but it may be readily separated from ammonia by the chloriodide of potassium and mercury, which precipitates it even more completely than tannic acid. It discharges the color of a solution of permanganate of potash more rapidly than ammonia, but more slowly than nicotina.

Organic Mixtures.—The process described for nicotina at p. 773, *post*, will be found effectual for the separation of this alkaloid from the contents of the stomach. It is easily separated by ether from its watery solution; and, on the addition of potash, the peculiar odor of conia is at once perceptible.

The reactions produced by tests on small quantities of an organic extract should be distrusted, unless there is strong corroborative evidence of the action of the poison on the body from the symptoms. As in reference to strychnia, veratria, and other alkaloids, an incautious operator may readily come to the conclusion that he has found "traces," and ascribe death to the poison. The following case occurred in Germany a few years since. A man died very suddenly, *i. e.*, in two hours and a half after going to bed, and it was alleged that his wife had poisoned him. The persons commissioned to make the analysis, deposed that they had found traces of conia in the stomach, intestines, and kidneys, and they came to the conclusion that the man had died from the effects of hemlock, which implicated his wife in a charge of murder. Some doubt appears to have arisen in the minds of the authorities on this point, and they submitted three questions for the consideration of a medical college. 1. Is there reason to believe that conia has really been found in the body of deceased? 2. If existing in the body, may it have been spontaneously produced, or does it show administration from without? Does its detection in the body incontestably prove that the deceased died from poisoning by conia or hemlock? 3. Is it improbable that deceased poisoned himself with hemlock? The college decided that there was not sufficient evidence to show that death had been caused by hemlock. The matter was then referred to Mitscherlich and Casper, of Berlin, and they found that the chemical processes pursued failed to detect conia in the body; that there was nothing to indicate that deceased had taken hemlock or conia in any form, and that the state of the windpipe sufficiently accounted for the sudden death of deceased. He had eaten and drunk freely, had vomited after going to bed; and a portion of the food had entered the trachea and had suffocated him. (See Casper's *Vierteljahrsschrift*, 1859, p. 194.)

WATER-HEMLOCK (CICUTA VIROSA).

The water-hemlock or *Cowbane* (Hooker) has given rise to several fatal accidents—its roots having been mistaken for parsnips. The whole of the plant is poisonous; but the roots are the most active, especially when gathered early or late in the year.

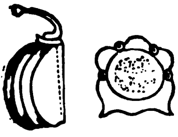
Symptoms and Effects.—The symptoms produced by the roots are giddiness, dimness of sight, headache, and difficulty of breathing. There is burning pain in the stomach, with vomiting, and these symptoms are accompanied by heat and dryness of the throat. Convulsions have been observed to precede death. In the cases of three children who died in convulsions from this poison, Mertzdorff found an injected state of the mucous membrane of the stomach, with redness of the air-passages, as well as of the cardia and pylorus; the vessels of the brain and the sinuses were filled with dark liquid blood. (Wibmer, *Cicuta*, 119.) In a fatal case which occurred to Wepfer, the patient, a man, æt. 20, who had eaten a large quantity of the *root*, was found with his face swollen and his eyes projecting. He breathed with great difficulty, and foamed at the mouth. He was seized with a severe epileptic fit; his limbs assumed a tetanic stiffness, and there was spasmodic breathing. He was quite unconscious, and speedily died. The only marked appearances were fluidity of the blood, and patches of redness on the mucous membrane of the stomach. (Wibmer, *loc. cit.*)

Dr. Badgely communicated some cases of poisoning by this plant to the *Montreal Medical Gazette* (June, 1844). Four children, between five and seven years of age, ate the roots of water-hemlock by mistake for parsnips. Within half an hour they were all seized with extreme nausea, burning pain at the pit of the stomach, and colicky pains in the bowels; they all complained, on reaching their homes, of sickness, for which warm milk was administered to them. Efforts to vomit were induced; in one, there was full vomiting, but in the other three nothing was ejected from the stomach. The pains gradually increased in two of them; and, in the space of about two hours from the time of their eating the roots, they were laboring under complete coma, with tetanic convulsions—the jaws were rigidly fixed, there was profound stertor, and the whole face was puffed and bloated, having precisely the appearance of the head of a person who had been for some hours under water; pulse intermitting, sometimes imperceptible. Emetics were exhibited, but without effect; and injections of castor oil and oil of turpentine were employed with great relief. The child who had eaten most sparingly had taken warm milk, and had vomited freely. One died in three hours; the others recovered.

A girl, æt. 8, who had eaten this plant, was found lying quite insensible. Her respiration was feeble, and rattling; the pulse soft, small, and scarcely perceptible; the pupils were dilated and fixed; the face pallid; limbs flaccid; abdomen distended; and there was general coldness of the surface, with an entire loss of the power of swallowing. Stimulating embrocations and cataplasms were applied, and after some hours the pupils contracted; the body became warm; the breathing easier; but there were involuntary movements of the limbs. There was a slight return of consciousness and the power of speaking, but the difficulty of swallowing continued; and the patient died in about sixteen hours. (Dr. Schlesier in Canstatt's *Jahresb.*, 1844, p. 229.)

A man ate a portion of the root of this plant in a cooked state. It had a sweetish taste, and was of the color of a parsnip. Half an hour after his dinner he felt giddiness and great dryness of the throat. He walked home with great difficulty, his legs being very unsteady, and all surrounding objects appeared to him as if they were advancing or receding. In about an hour and a half the legs were paralyzed; the arms numbed, and their movements weak; the face was anxious and flushed, and he had an apprehension of death. The skin was warm and dry; the pulse 90. An emetic was given. In two hours he was able to stand, and with difficulty walked across the room. He passed much urine, and had hallucinations. In seven hours the legs were cold, pupils dilated, skin and throat dry, with occasional delirium. There was no purging. In two days he recovered. (*Lancet*, 1871, vol. 2, p. 396.) In the *Pharmaceutical Journal* for June, 1872, p. 1063, two fatal cases are reported to have occurred at Chester. The boys ate the roots, supposing them to be wild celery. Symptoms of poisoning soon came on. They suffered from severe convulsions with trismus (lockjaw) before death.

FIG. 81.



Seeds of cicuta virosa, magnified (Lindley).

Analysis.—There are no means of identifying this plant except by the determination of its botanical characters. It grows abundantly on the borders of ditches, ponds, and streams. Its stem is thick, round, sparingly branched, and often attains four feet in height. At the lower part it is large, hollow, and divided by transverse partitions into large cells. The leaves are large, pinnated, and serrated; they have the taste of parsley. The root, which has a strong disagreeable smell and an acrid taste, is thick, short, hollow, and has numerous fibres at the joints. The nature of the poisonous principle is unknown.

The *Cicuta maculata* is possessed of equally virulent properties. Many fatal cases have occurred in the United States from the root having been eaten by mistake.

HEMLOCK WATER-DROPWORT (ŒNANTHE CROCATA).

This umbelliferous plant grows on the banks of rivers, streams, and ditches. It is one of the most poisonous of the order, and it is considered to be one of the most virulent of English vegetable poisons. It is found growing abundantly in various parts of England, and in the south of Ireland. Dr. Pickells has collected thirty cases of death from the eating of the root; the quantity taken in one instance did not exceed the top of the finger in size. The symptoms were insensibility, tetanus, delirium, and insanity. Sir R. Christison considers that this plant, as it grows in Scotland, is not poisonous; but there appears to be no doubt, from various recorded cases, that, as it grows in England, Wales, and Ireland, it is endowed with highly noxious properties.

Symptoms and Appearances.—A set of cases of poisoning by the œnanthe was communicated to the *Medical Gazette* (vol. 34, p. 288), by Dr. Bossey. A number of convicts, while engaged at work, at Woolwich, ate the leaves and roots of the œnanthe. In about twenty minutes, one man, without any apparent warning, fell down in strong convulsions, which soon ceased, but left a wild expression on his countenance. Soon afterwards, as many as nine fell into a state of convulsion and insensibility. The face of the man first seized became bloated and livid; there was a bloody foam about the mouth and nostrils; the breathing was stertorous and convulsive; there was great prostration of strength, and insensibility: he died in *five minutes* after the symptoms had set in. A second died under similar symptoms in a quarter of an hour, although the stomach-pump was used, and some leaves were extracted with the fluids. A third, who had assisted in carrying the two former, was himself seized with convulsions, and died in about an hour; and soon after him, a fourth died, in spite of the most energetic remedial treatment, by cold affusion, emetics, stimulants, stimulating friction, as well as the use of the stomach-pump. Two other cases proved fatal—the one in nine days, and the other in eleven; and in these two cases there was irritation of the alimentary canal. On inspecting the bodies of those who died quickly, there was congestion of the cerebral vesssls; and, in one instance, a layer of extravasated blood was found beneath the inner membrane (pia mater). In the first case, which proved most quickly fatal, the cerebral vessels were not congested. The pharynx and gullet had a white appearance, and contained some mucus, with portions of the root. The lining membrane of the windpipe and air-tubes was intensely injected with dark blood. The lungs were gorged with fluid blood. The blood in the heart was very black and fluid. The stomach and intestines were externally of a pink color; the cavity of the stomach was lined with a thick viscid mucus, containing portions of the root. The mucous membrane was much corrugated, and the follicles were particularly enlarged. Similar appearances were met with in all. In the two protracted cases, the mucous membrane of the stomach and bowels was softened and thickened. It had a pink color externally, but no red appearance internally. The vessels of the brain were congested. In the other convicts who partook of the roots the symptoms were not so severe. Under the free use of purgatives a considerable quantity of the root was discharged, and in a few days the men recovered. These cases show that the œnanthe is a powerful poison. It destroys life with even greater rapidity than arsenic, for it here proved fatal to a strong healthy man in less than *one hour*. Chemists have not yet ascertained on what principle its active properties depend, but they appear to reside chiefly in the root.

In March, 1846, Dr. Unger met with the following cases. A woman dug up some roots which she supposed to be parsnips. They were dressed for dinner as usual in an earthen pot in which her food was commonly prepared. The woman, as well as her husband and two children, partook of them. Dr. Unger was suddenly called to see them in the evening, and found them apparently laboring under delirium tremens. They were in constant motion, talking incessantly, without knowing what they said, and fancying they saw objects which had no existence. They fought with each other, and were occasionally attacked with fits of convulsive laughter. The countenance was pale, the pupil dilated, the look vague, tongue clean, moist, and tremulous; and the pulse, which, owing to the incessant motion, was felt with great difficulty, appeared smaller, weaker, and slower than natural. The patients rejected everything that was offered to them, aud were obliged to be restrained by force. A neighbor who had eaten a small portion of the roots, suffered from giddiness and general uneasiness; she was, however, perfectly conscious, and refused to take any remedy. Emetics led ultimately to the rejection from the stomach of a large quantity of the undigested root. After this, the symptoms abated; and the next morning, with the exception of a sense of weight in the head, they had all recovered. It is remarkable that there was no purging. (*Gaz. des Hôpitaux*, Sept. 19th, 1846.)

The *root* is considered to be the most active part of the plant; it is of a yellowish-white color, and not unpleasant to the taste. A very small portion of it, unless speedily ejected from the stomach, will suffice to destroy life. The symptoms have been occasionally delayed in their appearance; but, as in Dr. Bossey's cases, when they have once commenced, they run on to a fatal termination with great rapidity.

Dr. Woodville relates that three men ate, or rather tasted of the root. One was soon afterwards seized with convulsions and died; two others suffered from nervous symptoms, including locked-jaw, and one of these died; a fourth had dizziness, and he slowly recovered. It is remarkable that there was no vomiting, nor any tendency to vomit. The following set of cases occurred in Ireland. Eight boys ate the plant for water-parsnip. In four or five hours the eldest became suddenly convulsed, and died; and before the next morning four others died. Of the remaining three, one was maniacal for several hours; the other lost his hair and nails; and the third escaped. (*Medical Botany*, vol. 4, p. 144.) They who have vomited at an early stage have generally recovered.

In Sept. 1853, four children ate some of the roots of the œnanthe, the quantity taken being equal in size to a man's thumb. This was at 2 P.M. Four hours afterwards, according to the report of Dr. Nevins, one of them, a boy, was perfectly insensible, and his face was livid and turgid. He had previously vomited blood; and bloody mucus oozed from his mouth on admission. There were violent convulsions affecting the flexor muscles. The trunk was powerfully bent forward, the hands clenched even after death, and the jaws were rigidly closed. The respiration was spasmodic. The pupils were at first contracted, but afterwards dilated; they acted very feebly under the stimulus of light. The pulse was almost imperceptible. This continued until death, which occurred twelve hours after the taking of the poison. There was no return of consciousness, and the spasmodic contractions of the muscles continued with slight intermissions as long as the boy lived. The other children recovered. One was insensible and convulsed; a third had only abdominal pain and no cerebral symptoms. The quantity eaten in the latter case was unknown. (Mr. Kesteven's Quarterly Report on Toxicology, April, 1852, p. 583, from *Association Journal*, of December 2, 1853.)

In April, 1857, two fatal cases occurred at West Boldon in Durham. Two laborers ate some of the root of the œnanthe. They were found soon afterwards lying insensible and speechless, their faces livid, tongues swollen and protruded, and there were convulsive movements of their teeth, frothy mucus with blood about their mouths, eyes full and projecting, pupils dilated, breathing stertorous and labored, with occasional general convulsions. They both died in *an hour and a half* from the time at which they were first discovered. On inspection, it was found that there had been bleeding from the ears; the abdomen was livid and swollen. The stomach contained a gruelly liquid with some of the partly digested roots; on removing this liquid, the membrane was found congested and softened. The lungs were engorged with dark liquid blood, and the blood contained in the heart was in a similar state. Mr. Boyle, to whom these cases occurred, forwarded to me a portion of the roots, and there was no doubt that they were the roots of the œnanthe crocata.

In April, 1875, Mr. Drinkwater, of Llangollen, referred to me the following cases of poisoning with this plant. Several children ate the roots or tubers of the plant thinking they were parsnips. One was found dead and cold in two hours from the time that he was last seen. The hands, fingers, and toes were contracted as if he had been convulsed. The body was found about half a mile from the spot where the roots had been eaten. The deceased had evidently vomited. All the vessels of the head were loaded with black venous blood; all the external part of the brain below the pia mater appeared quite black with blood. The lungs and heart were in the same congested state.

The kidneys were darker than usual. The other organs were healthy. The stomach presented no particular appearance. It contained about half a pint of the masticated root of the œnanthe. Another boy had walked only fifteen yards from the brook. He had then fallen on his face and vomited. A third child who had eaten the root, went home and had a cup of tea. He vomited, and recovered. I examined the root, and found it to have the usual characters of the œnanthe.

In February, 1858, some sailors who had been sent ashore from their ship, collected a quantity of the tubers of the œnanthe, and ate them. Those who suffered were for the most part suddenly seized with symptoms of a violent kind, which came on in from half an hour to an hour. Some of the men who had eaten the roots were enabled to move about and assist in relieving the others up to the time at which they were themselves attacked. The first man seized was found insensible, and his body immovably rigid; he was moaning and breathing stertorously; his countenance was livid, the eyes were fixed and the pupils dilated; a bloody froth escaped from the mouth. There was opisthotonos; the pulse was feeble, and the action of the heart scarcely perceptible; the lower jaw firmly locked, the tongue much injured, and slightly protruding; death took place quietly in about eight minutes. In another case, in spite of violent vomiting, the man was seized with convulsions, and after a succession of them, died in two hours. The roots had been for some time in his body before emetics were given. On an inspection of the first case, the skin was livid, the stomach empty, but the mucous membrane was highly congested, and there adhered to it a tough viscid mucus. On opening the abdomen, a pungent odor was perceived, compared to that of burnt celery seed. Some portions of the root were found in the lower part of the small intestines.

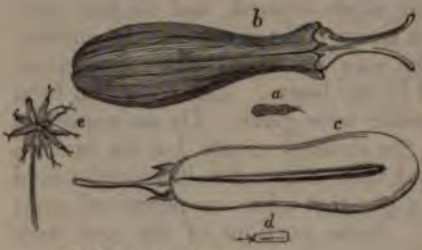
In addition to the convulsive symptoms above mentioned, the patients suffered from cramps in the legs, pain in the course of the spine, extending along the crural and sciatic nerves, giddiness, griping, eructations with the flavor of the root, debility, and total loss of appetite. (Dr. Grahame in *Med. Times and Gaz.*, March 6, 1858, p. 241.)

It is not often that attempts are made to destroy others by the administration of these vegetable poisons; but a case occurred in France, in which a woman attempted to poison her husband by mixing slices of the root of this plant with his soup. The woman was tried for the crime, and Mr. Toulmouche deposed at the trial that the plant from which the root had been taken, was the œnanthe crocata—that it was a powerful poison, and might cause death in two or three hours. The prisoner was convicted, and condemned to ten years at the galleys. (*Gaz. Méd.*, Jan. 3, 1846, p. 18; also, *Jour. de Chim. Med.*, 1845, p. 533.)

The *leaves* and *stems* of the œnanthe are very fatal to cattle. Dr. Cameron, of Dublin, met with the following case. A herd of seventy-four oxen were turned into the demesne of Lord Dunraven in April. In a few days the animals began to sicken, and in about a week, forty-three died! A veterinary surgeon who saw them stated that in most of the cases death took place soon after the illness was observed. The animals foamed at the mouth, had distended nostrils, shivered at the loins and the hinder extremities; the respiration was rapid and laborious, and they had tetanic spasms, the neck being curved laterally (pleurothotonos). Some of the animals reeled in a circle for several minutes, and then fell and died instantaneously. The stomachs of one were sent to Dr. Cameron for inspection. No ordinary poison was present, but in the herbage of the first stomach he found many fragments of a plant which he suspected to be œnanthe. He sent for some of the herbage of the demesne, and it included a large proportion of œnanthe crocata. There was no doubt that this was the cause of death. (*Lancet*, June 28, 1873, p. 918.)

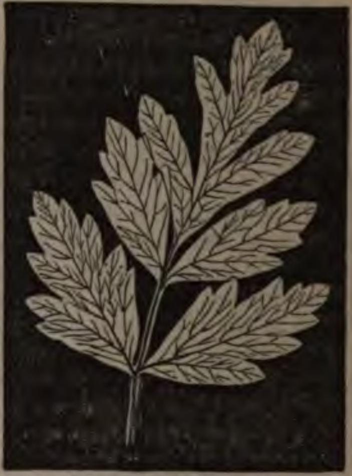
Analysis.—The œnanthe crocata can be identified only by its botanical characters. The leaves are of a dark-green color, with a reddish-colored border. They have no unpleasant odor when rubbed. The plant bears a greater resemblance to celery than most of the other umbelliferæ. Its stem is channelled, round, smooth and much branched, of a yellowish-red color, and growing to the height of three to five feet. The root consists of a series of large oblong fusiform tubes with long and slender fibres. When cut, it is of a yellowish-white color, and not unpleasant to the taste. It is the most active part of the plant. The leaves yield much tannic acid to water, but the decoction appears to contain no alkaloidal base, since the chloriodide of potassium and mercury produces no precipitate in it. The roots and stems of this plant are more frequently eaten than the leaves. Nevertheless, it may be occasionally necessary to identify the plant by the leaves. The above engraving (Fig. 83) is taken from a photograph of the larger leaves of the œnanthe crocata, grown from the roots of the plant procured from the spot where the two laborers above mentioned had taken their fatal meal. The smaller leaves of this plant are much wider in proportion to their length. No poisonous alkaloid has yet been separated from

FIG. 82.



Seeds of œnanthe crocata.
a. Natural size.
b. Magnified 30 diameters.
c. One half of the seed magnified.
d. One half natural size.
e. Group of seeds.

FIG. 83.



Leaves of œnanthe crocata.

the plant. The peculiar form of the seeds may aid identification (see Fig. 82, *supra*).

FINE-LEAVED WATER-HEMLOCK OR DROPWORT (ŒNANTHE PHELLANDRIUM OR PHELLANDRIUM AQUATICUM.)

This is another unbelliferous plant, which, like the œnanthe, is often popularly called water-parsnip. It grows by the banks of rivers, ditches, and ponds. It is poisonous, but less virulent than the œnanthe crocata.

Analysis.—The poisonous principle is unknown. The plant has a thick, hollow, smooth-jointed stalk, usually about three feet in height; the leaves are very fine, small, and much subdivided. They are of a dark shining green color; the root is thick, tapering, jointed, and sends off numerous long slender fibres.

FOOL'S PARSLEY (ÆTHUSA CYNAPIUM).

Fool's Parsley, or **Lesser Hemlock**, is very common in gardens and hedgerows. The leaves so closely resemble those of parsley that they have often been gathered for them by mistake.

Although this plant has been hitherto ranked among vegetable poisons, and described as such by Orfila, Wibmer, Sir R. Christison, Flandin, Galtier, and Lindley, the recent experiments of Dr. Harley lead to the conclusion that it is not possessed of poisonous properties. The alleged cases of poisoning with æthusa reported by different toxicologists he assigns to other vegetables mistaken for it, such as aconite, œnanthe, or hemlock. In cases of vegetable poisoning, botanical experts are rarely consulted, and mistakes respecting the real nature of a plant may be therefore easily made. Medical men who have reported cases of poisoning by the æthusa have certainly misdescribed the plant, and have thus raised a doubt on the trustworthiness of their reports. At the same time the symptoms which they have described, taken as a whole, are hardly consistent with the effects of aconite, œnanthe, or hemlock.

Symptoms and Appearances.—Lindley, in his *Medical Botany* (p. 248), describes the leaves of æthusa as poisonous, narcotico-acrid and emetic, and a frequent cause of dangerous accidents on account of their resemblance to parsley. A woman gave to two of her children some soup, in which she had boiled the *root* of this plant, mistaking it for the root of parsley. They were both seized with severe pain in the abdomen, and the next morning one of them, a boy, aged eight years, was in a state of perfect unconsciousness, and his jaws were spasmodically fixed. The abdomen was swollen; there was vomiting of bloody mucus, with obstinate purging; the extremities were cold, and the whole body was convulsed. He died in twenty-four hours. The only appearances met with were redness of the lining membrane of the gullet and windpipe, with slight congestion of the stomach and duodenum. (*Medicinisches Jahrbuch.*)

The *root* of the plant is also considered to have poisonous properties, and among the reported cases of poisoning with the root of æthusa are the following. In May, 1845, a girl aged five years, in good health, ate the bulbs (?) of the æthusa by mistake for young turnips. She was suddenly seized with pain in the abdomen, followed by sickness, but no vomiting. She complained of feeling very ill. On trying to eat, she could not swallow. She was incapable of answering questions, and her countenance bore a wild expression. The lower jaw became fixed, so as to prevent anything being introduced into the mouth. She then became insensible, and died in *an hour* from the commencement of the symptoms; so far as could be ascertained, there were no convulsions. A second child, aged three years, shortly after eating the same substance, was attacked with pain in the stomach, sickness, vomiting, and profuse perspiration. She soon recovered, with the exception of suffering severe griping pains without purging, but these disappeared the following day. A third child, of the same age, suffered from similar symptoms. Recovery in the two last cases was due to the plant having been eaten on a full stomach, and to the effect of early and copious vomiting. (*Med. Times*, Aug. 23, 1845, p. 408.) Mr. Thomas injected about two ounces of the juice expressed from the recent bulbs (?) into the stomach of a dog through an aperture in the gullet, which he afterwards secured by a ligature. There were violent spasms and urgent attempts to vomit. In most of the animals upon which this experiment was tried, death took place in from one to four hours. As Dr. Harley has pointed out, the root of the æthusa is not a bulb; nevertheless the account of the symptoms is hardly consistent with poisoning by œnanthe.

The following case occurred to Mr. Stevenson. Two ladies partook of some salad, into which æthusa cynapium had been put by mistake for parsley. They soon experienced a troublesome nausea, with occasional vomiting; oppressive headache, giddiness, and a strong propensity to sleep, at the same time that this was prevented by frequent startings and excessive agitation. There was a sensation of pungent heat in the mouth, throat, and stomach, with difficulty of swallowing, thirst, and loss of appetite. There was numbness, with tremors of the limbs. The two patients only slowly recovered from the effects of the poison. (Churchill's *Botany*.)

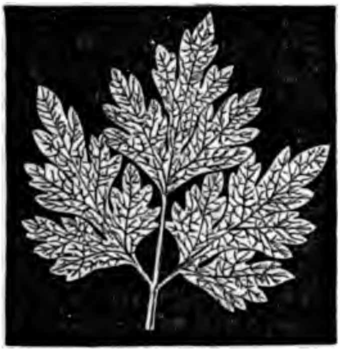
Dr. Harley, after quoting fifteen or sixteen reported instances of poisoning with æthusa cynapium, draws the conclusion that the plant is harmless, and ought to be expunged from the list of vegetable poisons. He performed a variety of experiments on himself and his patients with the juice of the plant and with tinctures prepared from the ripe and green fruit, with the fluid extract and the oleo-resin separated from the plant.

The largest dose of the juice given was four ounces of the spirituous mixture, which is equivalent to three ounces of the fresh juice and to six ounces of the fresh herb—a quantity greater than was assumed to have been taken in any of the alleged cases of poisoning. The maximum dose of the tincture of the ripe fruit was a fluid ounce, equivalent to ninety grains of the fruit. The largest dose of the tincture of the unripe fruit was two fluid ounces, equivalent to more than 300 grains of the fruit. Of the oleo-resin ten grains were given in solution to a healthy adult. With these doses no effects whatever indicative of a poisonous action were produced. No trace of gastric irritation or any symptom, immediate or subsequent, occurred in any case. (*St. Thomas's Hospital Reports*, 1873, vol. 4, p. 43.)

Analysis.—The æthusa is known from garden parsley by the smell of its leaves when rubbed, which is peculiar, disagreeable, and very different from that possessed by the leaves of parsley (see Fig. 79, p. 701, *ante*). The *leaves* of fool's parsley are finer, more acute, decurrent, of a darker green color. They are represented in the annexed illustration (Fig. 84) from a photograph of the leaf of the living plant.

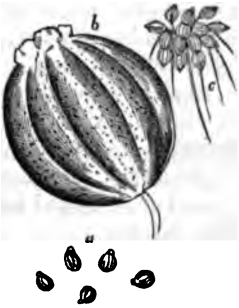
Fig. 85 represents the *seeds* of the plant, of the natural size and magnified. They differ from those of other umbelliferæ. The flower-stem, which is striated or slightly grooved, is easily known from all other umbelliferous plants by the beard, or three long pendulous leaves of the involucrum under the flower. The flowers are white; those of the garden parsley of a pale yellow color. The *root* is fusiform.

FIG. 84.



Portions of a leaf of fool's parsley, natural size.

FIG. 85.



Seeds of fool's parsley.

a. Natural size.
b. Magnified 30 diameters.
c. Group of seeds.

This plant was, at one time, supposed to contain conia, but neither this nor any other alkaloid has been separated from it.

lyzing the muscles of respiration, and causing asphyxia; and 3, by producing syncope. The last is the most common mode of death in man, when the case is protracted for some hours. A dose sufficiently large to cause death by syncope, excites in the first place, numbness and burning heat in the mouth, throat, and stomach; pain in the abdomen, with sickness and vomiting; diminished sensibility of the skin, giddiness, dimness of vision, or complete blindness, ringing in the ears, and occasionally deafness, frothing at the mouth, a sense of constriction in the throat, with sensations of weight and enlargement of various parts of the body, but especially of the face and ears, great muscular weakness, with general trembling, greater or less difficulty of breathing and speechlessness, sinking at the pit of the stomach, pulse small, feeble, irregular, finally imperceptible, extremities and surface of the body cold and clammy, countenance blanched, and the lips bloodless. The individual dies suddenly; the mental faculties are commonly retained to the last, or there is only slight delirium. The case generally proves fatal in from one to eight hours; if it last beyond this period, there is good hope of recovery. The most common appearance on inspection is a general congestion of the venous system. The brain and membranes are gorged; in some instances there is a redness of the mucous membrane of the alimentary canal. (*An Inquiry on the Properties of the Aconitum Napellus*, 1845, p. 43.)

Leaves.—In the *Lancet* for June 28, 1856 (p. 715), is reported the case of a child, between two and three years of age, who died in about twenty hours after eating some of the fresh leaves of aconite. The first symptoms were severe pain in the abdomen, vomiting, and a contracted state of the pupils; these were followed before death by collapse and coma. The stomach and intestines were found much inflamed, the latter presenting some patches approaching to gangrene. The leaves of aconite are of a dark green color, thick, and of a peculiar palinated shape. The annexed illustration (Fig. 86, p. 716) is engraved from a photograph of a fresh leaf. When masticated the leaves slowly produce on the lips and tongue a persistent sensation of tingling and numbness, with the sense of coolness observed in the root. They are less powerful than the root and seeds. The seeds differ in appearance from those of other poisonous plants. (See Fig. 87.)

A boy, æt. 14, ate some of the leaves for parsley. In about two hours he complained of a burning sensation in the mouth, throat, and stomach, and vomited freely. Soon after this he fell on the ground in a fit, and seven hours after having taken the poison he was found lying across the bed with his hands in his pockets, dead. On inspection, the cerebral vessels were filled enormously with dark-colored fluid blood, upwards of a pound of which escaped from the skull and spinal canal. The stomach was empty; there was a deep inflammatory blush over the whole mucous surface, with patches of a darker color. (*Med.-Chir. Rev.*, July, 1844, p. 261; see also case in *Lancet*, June 28, 1856, p. 715.)

A well-marked case of poisoning by a *decoction* of the fresh stalks and leaves of this plant occurred to Mr. Sayle. A man, æt. 39, boiled them in half a pint of beer until it was reduced to a quarter of a

CHAPTER LXXII.

CEREBRO-SPINAL POISONS—ACONITE OR MONKSHOOD—SYMPTOMS AND APPEARANCES—LEAVES—EXTRACT—ROOT—POISONING WITH THE TINCTURE—SYMPTOMS AND APPEARANCES—FLEMING'S TINCTURE—ANALYSIS: BOTANICAL AND CHEMICAL—ACONITINA.

MONKSHOOD (ACONITUM NAPELLUS).

THIS well-known garden plant is in some parts of the country called *Wolfsbane*, and in Ireland *Blue-rocket.* The roots, seeds, and leaves are highly poisonous, owing to the presence of the alkaloid *aconitina.* The *root* is especially noxious, and when the leaves have fallen off it appears to possess its greatest virulence. The root and leaves, when masticated, produce a cool numbing sensation, affecting the lips, tongue, and interior of the mouth generally. At first the root appears to be tasteless, as the effects are only fully manifested after twenty minutes or half an hour. From tasting only a small portion of the dried root, I found that this disagreeable sensation remained on the tongue and lips for four hours. In larger quantity the taste has been described as burning, and it is stated to have been followed by a hot acrid sensation in the throat.

Symptoms and Appearances.—In from five minutes to an hour after the poison has been taken, the patient complains of numbness and tingling in the mouth and throat, which are parched; there is headache and giddiness, with numbness, and tingling in the limbs, a loss of power in the legs, frothing at the mouth, severe pain in the abdomen, followed by vomiting and purging. In some cases the patient is completely paralyzed, but retains his consciousness; in others the giddiness is followed by dimness or loss of sight, delirium, and other cerebral symptoms, but not amounting to the complete coma produced by the cerebral or narcotic poisons. As the symptoms progress in severity, the patient complains of a general numbness and tingling in the fingers, arms, and legs. There is great pain or a sense of weight in the region of the heart. The pulse is small, fluttering, and irregular, sometimes almost imperceptible; the heart's action is weak, and, as observed in two cases reported by Dr. St. Clair Gray, intermittent. He also noticed in these cases a dragging of the limbs and skin. The eyes are generally brilliant and staring, the pupils widely dilated, the skin cold and livid, the breathing difficult, with a feeling as if the breathing would be entirely suspended. Convulsions are not commonly observed in man, or they are indicated by general tremors, and twitchings or cramps of the voluntary muscles. The poison produces convulsions in animals.

Dr. Fleming, who has closely investigated the subject of poisoning by aconite, considers that this poison may cause death—1, by producing a powerfully sedative impression on the nervous system; 2, by paralyzing the muscles of respiration, and causing asphyxia; and 3, by producing syncope. The last is the most common mode of death in man, when the case is protracted for some hours. A dose sufficiently large to cause death by syncope, excites in the first place, numbness and burning heat in the mouth, throat, and stomach; pain in the abdomen, with sickness and vomiting; diminished sensibility of the skin, giddiness, dimness of vision, or complete blindness, ringing in the ears, and occasionally deafness, frothing at the mouth, a sense of constriction in the throat, with sensations of weight and enlargement of various parts of the body, but especially of the face and ears, great muscular weakness, with general trembling, greater or less difficulty of breathing and speechlessness, sinking at the pit of the stomach, pulse small, feeble, irregular, finally imperceptible, extremities and surface of the body cold and clammy, countenance blanched, and the lips bloodless. The individual dies suddenly; the mental faculties are commonly retained to the last, or there is only slight delirium. The case generally proves fatal in from one to eight hours; if it last beyond this period, there is good hope of recovery. The most common appearance on inspection is a general congestion of the venous system. The brain and membranes are gorged; in some instances there is a redness of the mucous membrane of the alimentary canal. (*An Inquiry on the Properties of the Aconitum Napellus*, 1845, p. 43.)

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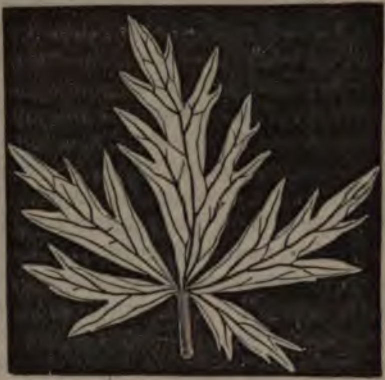
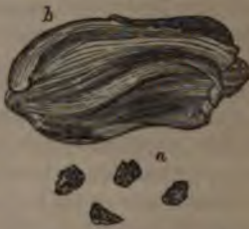
A well-marked case of poisoning by a *decoction* of the fresh stalks and leaves of this plant occurred to Mr. Sayle. A man, æt. 39, boiled them in half a pint of beer until it was reduced to a quarter of a pint; he then swallowed half of it as a medicine. An hour afterwards he was found in bed, rolling his arms about and foaming at the mouth; the pupils were widely dilated, the legs were paralyzed, the skin was cold and clammy, there was great nausea, the pulse was scarcely perceptible, and he was perfectly insensible. He soon afterwards died.

FIG. 86.



Small leaf of aconite, natural size, from a photograph.

FIG. 87.



a. Seed of aconite, natural size.
b. The same, magnified 30 diameters.

The abdomen was examined, and the only appearance met with was a slight redness near the cardiac extremity of the stomach. (*Med. Times*, Oct. 18, 1845, p. 70.)

Extract.—This is made from the leaves of the plant. It varies in strength, according to the care used in its preparation. In the hospital at Bordeaux, *five grains* of fresh extract of aconite were given to three patients. One of them died in three hours. In a quarter of an hour after taking the poison, the patients had tremors of the muscles, and a pricking sensation over their bodies; severe vomiting followed. They became quite unconscious, and on recovering their senses there was confusion of sight, with intense headache; the skin was cold and clammy, the pulse slow and irregular, and the breathing short and hurried. Two of the patients recovered. (*Med.-Chir. Rev.*, Oct. 1839, p. 544.) In November, 1862, a case was communicated to me by Dr. Vachell, of Cardiff, in which a man died from the effects of two grains of the extract of aconite taken in two pills. As in other cases in which active poisons have been administered in pills, the symptoms were a long time in appearing, but when they once commenced, they proceeded rapidly to a fatal termination.

Root.—Poisoning by the *root* of aconite is by no means unfrequent. In the autumn, the root is liable to be mistaken for that of horseradish. It has been thus accidentally eaten on several occasions, and has caused death. It produces, when eaten, a sense of tingling and numbness in the lips, with a burning sensation in the mouth and throat, extending to the stomach. (Pereira, *Mat. Med.*, vol. 2, pt. 2, p. 688.) A fatal case arising from the root having been eaten by mistake for horseradish, occurred at Bristol in the autumn of 1853. The deceased in this case is stated to have taken only as much as would go on the point of a table-knife. Mr. Herapath calculated the quantity at thirty-five grains, and estimated it to be equivalent to one-twentieth of a grain of pure aconitina. A similar mistake led to fatal results in three hours in a case which occurred at Lambeth, and another set of cases occurred at Dingwall, in Scotland, in January, 1856. Here three persons were poisoned by reason of their having had sauce, made with the root of aconite, served at dinner with roast beef in place of horseradish sauce! They were healthy adults; they all died within three hours and a half. Mistakes of this kind show deplorable ignorance, but there is always the risk of their occurrence when horseradish and aconite are grown near to each other in a garden, at that season of the year when the leaves have fallen.

A trial for murder by poisoning with the root of this plant took place at the Monaghan Lent Assizes in 1841 (*Reg.* v. *McConkey*), in which the late Dr. Geoghegan, of Dublin, conducted the medico-legal investigation. The medical evidence was beset with difficulties, for no trace of the poison could be discovered in the body, and it was only by a close analysis of symptoms and appearances that the charge was

FIG. 88.
a

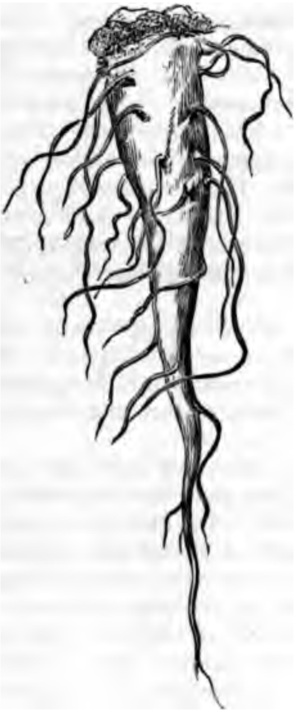
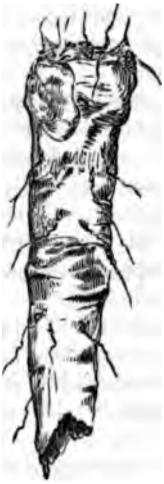


FIG. 89.
b



a. Root of aconite. *b*. A portion of the root of horseradish.

brought home to the prisoner. The deceased had eaten for his dinner some greens dressed for him by the prisoner; he complained of their having a sharp taste, and this was perceived also by another person present, who tasted them. It was ascertained that the deceased, soon after the meal, had vomited a greenish matter, and suffered from purging, restlessness, incoherence, lockjaw, and clenching of the hands. He died in about three hours after having eaten the greens, but was not seen by a medical man while living. The chief appearance met with was in the stomach, where the mucous membrane was of a light reddish-brown color. Traces of vegetable matter were found in the intestines; but no poison could be detected either botanically or chemically. The symptoms suffered by a friend of the deceased, who had accidentally tasted the greens, were very characteristic of poisoning by aconite. In *two* minutes he felt a burning heat in the mouth, throat, gullet and stomach, then a sensation of swelling in the face, with a general feeling of numbness and creeping of the skin. Restlessness, dimness of sight, and stupor almost amounting to insensibility, followed, and in about an hour after the meal he was found speechless, frothing at the nose and mouth, the hands and jaws clenched, appearing occasionally as if dead, and then again reviving. Vomiting, purging, tenderness at the pit of the stomach, cramps, tingling of the flesh, and a burning taste in the mouth, followed. This man did not entirely recover until after the lapse of five weeks. The prisoner was convicted of murder, and confessed before her execution that the powdered *root* of aconite had been mixed with pepper and sprinkled over the greens. (*Dub. Med. Jour.*, vol. 19, p. 403.) The non-discovery of the vegetable or of the alkaloid aconitina in the body was raised as an objection to the opinion of Dr. Geoghegan in the case of McConkey, but the medical and general evidence taken together was considered to be conclusive of the fact of poisoning.

Dr. Geoghegan, in the paper referred to, quotes two other instances of poisoning with aconite, one of a man aged fifty-six, who died in an hour and a quarter after eating the root; and the second, a boy aged seven, who died in two hours, having been much convulsed before death.

It is stated that one drachm of the dried root has been known to prove fatal; but it is probable that less than this would cause death. In November, 1856, Mr. Hadfield forwarded to me four small slices of the root, taken from the stomach of a man who died in three hours. The quantity which he had swallowed with suicidal intention was unknown; but none was thrown off by vomiting so far as could be ascertained. The *symptoms* within half an hour of death were burning pain in the stomach, parched mouth, intense thirst, retching and vomiting of a tenacious mucus, cold perspiring skin, imperceptible pulse, and a feeling of deadly sickness. The patient was conscious; there were no convulsions. On inspection, there was congestion of the brain as well as of its membranes; the heart was flaccid; it contained some blood on the right side. The stomach contained much half-digested food, with four slices of aconite root, apparently unaltered. The mucous membrane presented a slight reddish-brown patch at the greater end, of the size of half a crown. It was otherwise healthy, as well as the other organs. (For a further account of poisoning by this plant, I must refer the reader to a paper by Dr. Geoghegan, *Dub. Jour. Med. Sci.*, vol. 19, p. 403.)

Aconite finds a place among Indian poisons. From a statistical list furnished to me by Dr. B. Brown, there were nineteen cases of poisoning by aconite in the Punjab alone, in the years 1861–73.

Analysis.—There are these striking differences between the roots: 1. Aconite root (Fig. 88) is very short, conical, and tapers rapidly to a point. 2. It is externally of an earthy brown color—internally white and of an earthy smell—the cut surface is rapidly reddened by exposure to air. It has numerous long thin fibres proceeding from it. 3. It has at first a bitter taste, but after a few minutes it produces a disagreeable sense of tingling and numbness on the lips and tongue. 1. Horseradish root (Fig. 89) is long, cylindrical, or nearly so, and of the same thickness for many inches. 2. It is externally whitish-yellow, and has a pungent odor when scraped. 3. Its taste is sometimes bitter, but it produces an immediate hot or pungent sensation, without any feeling of numbness.

The Tincture—Symptoms and Appearances.—There are numerous instances recorded of poisoning by aconite under the form of tincture of the root. In a case which occurred to M. Devay (*Cormack's Edinburgh Journal*, April, 1844), a man is stated to have recovered in three days after having taken upwards of ten drachms of the tincture (only infused for a day); but this could have contained no aconitina. The late Dr. Male, of Birmingham, died from the effects of not more than *eighty drops* taken in ten doses, over a period of four days, the largest quantity taken at once being *ten* drops. (*Prov. Med. and Surg. Jour.*, Aug. 20, 1845, p. 535; also *Med. Gaz.*, vol. 36, p. 861.) The late Dr. Pereira informed me that he had known tingling and general numbness of the limbs produced in hysterical females by a dose of only *five minims* of a carefully prepared tincture. Dr. Topham has published an account of the symptoms produced by *fifteen minims* of the tincture of the root of aconite. Immediately after taking the poison in a mixture into which it was put by mistake, the patient (a woman æt. 27) felt a sensation of numbness in the tongue, with difficulty of swallowing. There were convulsive twitchings of the muscles of the face, and she lost the power of walking. There was complete unconsciousness, which continued for two hours, when she began to recover. The pupils were observed to be slightly contracted. The intensity of the symptoms varied at intervals, and came on in paroxysms. They indicated great disorder of the nervous system. The next day she had numbness in both arms, but she rapidly and perfectly recovered. (*Lancet*, July 19, 1851, p. 56. See also a report of a case of recovery in *Amer. Jour. Med. Sci.*, Jan. 1862, p. 285.)

The tincture varies much in strength. In the *Lancet*, vol. 2, 1861, p. 170, it is stated that a lady recovered who had swallowed two teaspoonfuls by mistake for laudanum. She had been in the habit of taking large doses of laudanum. After she had swallowed the aconite she could not rise from her seat, and exclaimed that she had lost the use of her legs. She complained of a burning sensation in the throat and constriction at the chest. Her mind was clear, and she had the consciousness of no feeling in her arms and legs. The symptoms subsided in two hours, and she recovered in eight hours. Vomiting had been early promoted by emetics. When the tincture has been taken, the effect upon the lips and tongue will furnish valuable evidence, as this property is peculiar to aconite. Taken together with the symptoms, it will be strong evidence of the nature of the poison. At the trial of Dr. Pritchard, for the murder of his wife and mother-in-law at Glasgow in 1865, Drs. Penny and Adams inferred that the poison administered was aconite by the sense of taste. The prisoner had artfully added the tincture of aconite to Battley's Sedative Solution, which the wife's mother had been in the habit of taking. A bottle, with a portion of the "sedative" remaining in it, was found in the pocket of the deceased. A soft extract was obtained by evaporation, which, when applied to the lips, had the tingling and benumbing taste of a similar extract obtained from Fleming's tincture of aconite. He obtained from it aconitina by Stas's process, and by physiological experiments on rabbits he proved that aconite was the cause of death. (*On the Detection of Aconite*, Glasgow, 1865.)

In January, 1853, a case of poisoning by tincture of aconite occurred at a convent near Bristol. One of the inmates named "Forty" had taken, by mistake, *seventy minims* of *Fleming's tincture* of the root mixed with one grain of acetate of morphia. This was about seven o'clock in the morning. In a few minutes she became very thirsty, complained of a burning sensation and pain in her stomach, to relieve which she swallowed a quantity of cold water. In fifteen minutes there was violent vomiting, which continued for two hours. She lost the power of standing, and was very restless. The pain in the stomach increased. After the first hour she was unable to do more than turn her head and vomit. There was violent straining as well as convulsive movements of the muscles. At nine o'clock she had a stupefied look, complained of giddiness, and was covered with a cold sweat. At ten o'clock she was quiet as if asleep. She was conscious until shortly before her death, which took place in about four hours after she had taken the poison. There were no general convulsions; the pain in the stomach was well marked throughout. On inspection, the face and lips were found swollen and dark-colored, eyes bright, pupils dilated, and the muscular system rigid. The membranes of the brain were congested, but the brain itself was firm and healthy; the lungs were healthy; there was merely cadaveric congestion from gravitation. The heart was flaccid, uterus congested, bladder empty, and sphincter ani relaxed. The stomach contained some mucus, and the membrane at the larger curvature was injected (reddened) in patches, but otherwise natural. The mucous membrane of the duodenum was in a high state of inflammation, abraded in patches, softened, and broken down. Some spots were of a very dark color, passing to mortification. It is proper to observe that the deceased died on the 5th January, and the inspection was not made until the 14th. (Report by Dr. O'Bryen, *Association Med. Jour.*, Jan. 28, 1853, p. 92.) In a case reported in the *Lancet*, 1855, vol. 1, p. 467, a woman died in five hours from two drachms of the tincture taken with suicidal intention. There were no narcotic symptoms.

In 1853 a healthy young man lost his life at Glasgow, by reason of his having taken a mixture containing *twenty-five minims* of tincture of aconite, twenty minims of tincture of belladonna, and a drachm of the tincture of musk. The tincture in this case was prepared with sixteen ounces of the root of aconite to thirty fluid ounces of spirit. The mixture was swallowed at 6.30; the patient walked to a friend's house about three-quarters of a mile distant, which he reached at 7.20. He then complained of being sick, and of a tingling sensation in his hands and arms. In a short time his hands and arms were so benumbed and powerless, that when he raised them he could not keep them up. Vomiting came on, with convulsive movements of the body, the pulse could not be felt, and the patient, retaining his consciousness to the last, died within three hours from the time of taking the poison. The body was inspected two days after death by Dr. Easton. The veins of the brain were unusually congested, and there was a great quantity of serum effused in the arachnoid membrane. The lungs and the right cavities of the heart were gorged with dark blood. The lining membrane of the stomach was of a dark-red color. Death was very properly referred by Dr. Easton to the action of aconite. (*Assoc. Med. Jour.*, Sept. 16, 1853, p. 817.)

The case of the man Hunt, who, in November, 1863, destroyed his wife and children by prussic acid, presents some features of interest in reference to the symptoms and appearances produced by tincture of aconite. The quantity of tincture taken by him was not determined; but the man was soon afterwards seized with violent spasmodic retching, face pale, skin cold and clammy, pulse small and hardly perceptible, and the action of the heart feeble. The pupils were much dilated, and the eyes brilliant and sparkling; the breathing quiet and regular, except during the fits. He complained of pain in the heart. In attempting to walk he staggered, and had no power to raise his arms. He was perfectly conscious, called for writing materials, and wrote a few lines. He then became suddenly worse, and a quarter of an hour before his death he lost all power and sensation in his limbs, the sharpest pinches producing no impression. The pulse was imperceptible. There were no convulsions, but complete relaxation of the limbs at death, which appeared to arise from syncope three-quarters of an hour after he had taken the poison. On inspection forty-two hours after death, there was great rigidity of the muscles. The substance of the brain was firm and healthy; the vessels on the surface were filled with blood. The heart was healthy; the right side was greatly distended with dark fluid blood; the left side contracted and quite empty. The lungs were healthy. In the abdomen the viscera were healthy, with the exception of the stomach and duodenum. Mr. Puckle, to whom I am indebted for the above particulars, brought the stomach to Guy's Hospital, and we examined it together. There was great capillary congestion at the larger end of the stomach, the mucous membrane having a bright-red color. There were marks of irritation, with softening and separation of the mucous lining, the whole of the membrane being in a highly corrugated condition. Traces of aconitina were found in the contents of the stomach. The deceased had provided himself with an ounce of the tincture of aconite, and had swallowed the greater part of this mixed with water.

Several cases of poisoning occurred some years since at Lille, in which tincture of the fresh root of aconite was taken by mistake for a cordial. The symptoms appeared in three members of a family in half an hour; there was severe burning pain in the throat and stomach, with vomiting, purging, and tenderness of the abdomen. One died in two hours; the second in two and a half hours; the third, who had delirium, recovered. The only appearance met with on inspection was great redness of the mucous membrane of the stomach and small intestines. (*Ed. M.* 452.)

In February, 1 died from the effects of *one drachm* of the tinc . The symptoms were giddiness, with intense burning tongue to the throat, a loss of power in the legs, coldness and feet and along the spine, twitchings of the musc rs, and toes, dimness of vision, dilatation of the pupil low, weak pulse, and involuntary evacuations. Just was one universal convulsion. He revived, gasped, and is skin at this time was cold, his lips were blue, his pulse perceptible, and his breathing oppressed. He died an hour after he had taken the poison, which had been dispensed by mistake for another tincture. Emetics produced active vomiting, but the poison had already become absorbed. On inspection, the stomach contained a dark-brown fluid; the mucous membrane was congested in patches of various sizes, and of a dark-red color. The right side of the heart, which was healthy, was filled with liquid venous blood. The liver, spleen, kidneys, and intestines were congested. The bladder was empty. (Dr. Bone in *Lancet,* April 5, 1856, p. 369.) In September, 1857, the wife of a physician, of Durham, died from the effects of one drachm of tincture of aconite given in two doses, at an interval of some hours. It had been given by mistake for tincture of henbane.

The tincture of aconite, according to the British Pharmacopœia, is made by macerating the *root* in rectified spirit. Fleming's tincture is also made with the root, but with half the quantity of spirit. The *medicinal dose* of this tincture is variously stated, owing to the great difference in its strength. The dose of the B. P. tincture is from five to fifteen minims. The late Dr. Pereira states that a dose of six minims administered twice, produced the most alarming symptoms in a healthy young man. (*Mat. Med.*, vol. 2, part 2, p. 693.)

Fleming's tincture is a powerful preparation, and might, from its appearance, be easily mistaken for sherry wine. Since this tincture is as deadly in its operation as prussic acid, and so many accidents have occurred from the use of it, it seems advisable that its strength should be reduced. In October, 1852, an excise officer lost his life by merely tasting Fleming's tincture of aconite, under the supposition that it was flavored spirit. He was able to walk from the Custom House over

London Bridge, but he died in about four hours after taking the poison. A liquid sold for external use under the name of NEURALINE appears to be a preparation of tincture of aconite mixed with chloroform and rose-water. According to Dr. Harley, there is one drop and a half of Fleming's tincture in half a bottle of the so-called neuraline. It operates by causing numbness or paralysis of the parts to which it is applied. The death of the Hon. G. R. Vernon was ascribed to the too frequent use of this preparation externally. (*Pharm. Jour.*, Jan. 1872, p. 618.) The same ignorance prevails respecting this as with regard to other poisonous substances dissolved in alcohol, namely, that it is harmless unless the skin is broken. Unless it were absorbed by the skin it could have no medicinal operation, and the effects of absorption must depend on the quantity applied and the frequency with which it is applied. Alcohol has been found to promote the absorption of poisonous agents through the unbroken skin. (See p. 25, *ante.*)

Analysis.—The botanical characters of the leaves and root of this plant, when any portions can be obtained for examination, will enable a medical witness to identify it. The root has been frequently and fatally taken for horseradish, but there are striking differences.

ACONITINA.

The alkaloidal base of this plant, *aconitina* or *aconitia,* is a most formidable poison, exceeding all others in its effects. The proportion of *aconitina* contained in the fresh root, amounts to about a quarter of a grain in an ounce, but it is subject to variation. According to Mr. Herapath, the dried aconite root grown in England contains from twelve to thirty-six grains in the pound. The roots after flowering contain the largest proportion. According to the late Dr. Pereira, it is strongly retained in the vegetable tissues even after their compression. Hence, the uncertainty of the strength of the preparations of aconite. Although there are few poisons so deadly as aconitina—for even experiments on it require to be made with the greatest caution—a singular instance is recorded by the late Dr. G. Bird in which a gentleman is stated to have recovered after having taken *two grains and a half.* (*Med. Gaz.*, vol. 41, p. 30.) In this case, however, there appears to have been early and copious vomiting, so that the greater part of the poison had probably been discharged. Enough had been absorbed, however, to produce most serious symptoms. There was collapse, coldness of skin, cold perspiration, the heart's action was scarcely perceptible, and there was constant spasmodic vomiting of a violent kind.

The late Dr. Pereira states that this alkaloid cannot be administered internally with safety. In one case one-fiftieth part of a grain nearly proved fatal to an elderly lady (*Mat. Med.*, vol. 2, part 2, p. 695); and it is probable that *one-tenth* part of a grain of pure aconitina would prove fatal to an adult. It would seem, however, that some samples of this alkaloid are much less potent than others, and the chemical properties are also different. (See paper by Schroff, Reil's *Journal für Toxikologie*, H. 3, 1857, p. 335.) The case reported by the late Dr. Golding Bird, *supra,* may thus receive an explanation. It appears that some samples of English aconitina are more potent than those of

Germany, but according to Dragendorff the difference in the poisonous effects depends not on the relative proportion of aconitina, but on the presence of another alkaloid, which he describes under the name of *Nepaline*. Thus the most powerful of the species is the *Aconitina ferox*, and of this nepaline is a constituent. When this plant is used for making the medicinal preparations of aconite these are observed to have a greater potency. The action of Nepaline is different from that of aconitina in similar doses. An animal is so soon killed by nepaline or by the aconitina ferox in any form that the poison does not reach the large intestines, and it has never been detected in the urine. (*Toxicologie*, 1873, p. 301.) The only pharmaceutical preparation of this alkaloid is an ointment (*unguentum aconitiæ*). It consists of eight grains of aconitina dissolved in spirit and mixed with one ounce of lard.

Chemical Properties.—Aconitina is not very soluble in water, but it is dissolved by four parts of alcohol, two of ether, and two and a half of chloroform. It is soluble in benzole. A sample of Morson's aconitina possessed the following properties: It was in whitish granular masses, without any distinctly crystalline structure. 1. When heated it readily fused and burnt in the air with a bright-yellow flame. 2. Heated in a close tube, it evolved first an alkaline and then an acid vapor. 3. It was scarcely soluble in water, but was dissolved by weak acids and alcohol; it did not form a crystallizable salt on evaporation. 4. Nitric acid dissolved it without causing any change of color. 5. Sulphuric acid produced no change of color, but on adding a crystal of bichromate of potash, green oxide of chromium was set free. 6. Iodic acid dissolved it without change of color. 7. Sulphomolybdic acid produced with it a pale yellowish color—sometimes produced by sulphuric acid only. 8. Its solutions were precipitated by tannic acid, and the chloriodide of potassium and mercury. 9. Iodine-water gave a reddish-brown precipitate in a solution of the sulphate. 10. It was precipitated whitish-yellow by chloride of gold, but not by chloride of platinum. Gallic acid, corrosive sublimate, iodide and sulphocyanide of potassium produced no change in the solution. Dragendorff recommends concentrated sulphuric acid as a good test. It dissolves the alkaloid and acquires a yellow color, which becomes brown, then reddish-brown and violet. The violet color shows itself first on the edges and is the more slowly produced as the aconitina is in larger quantity. The change of color takes place in about two hours with a small quantity. The bihydrate and a diluted acid is without effect. (*Op. cit.*, p. 393.)

Organic Liquids.—This alkaloid is sufficiently soluble in ether to be separated by Stas's process employed for strychnia (*ante*, p. 689). Dr. Headland has recommended as a physiological test for aconitina, the application of an alcoholic extract of the acid contents of the stomach. If $\frac{1}{20}$th of a grain be obtained it will be sufficient. He states that $\frac{1}{300}$th of a grain will poison a mouse with characteristic symptoms; $\frac{1}{100}$th a small bird; $\frac{1}{1000}$th of a grain causes tingling and numbness of the tip of the tongue; $\frac{1}{100}$th dissolved in spirit and rubbed into the skin causes loss of feeling, lasting for some time. (*Lancet*, March 29, 1856, p. 343.) There is a great difference in the chemical and physiological properties of this alkaloid, according to the mode in which it is prepared. (Bouchardat, *Ann. de Thérap.*, 1864, pp. 48 and 54; also 1863, p. 41.) In order to separate aconitina from the root or leaves Messrs. Boiroux and Léger advise: 1. Making a boiling decoction in water containing one per cent. of tartaric acid. 2. Evaporation to a syrup, and the residue rendered strongly alkaline with caustic soda. 3. The alkaline liquid poured a little at a time into benzole. 4. The benzole shaken with a little water acidulated with tartaric acid. 5. Precipitation of the aconitina from the aqueous acid solution by ammonia. (*Pharm. Jour.*, Feb. 1875, p. 662.)

CHAPTER LXXIII.

BELLADONNA OR DEADLY NIGHTSHADE—SYMPTOMS—ACTION OF THE ROOT, LEAVES, AND EXTRACT—LOCAL ACTION—APPEARANCES—ANALYSIS: MICROSCOPICAL, CHEMICAL, AND PHYSIOLOGICAL—ATROPIA—ITS ACTION IN THE BODY—CHEMICAL AND PHYSIOLOGICAL PROPERTIES.

DEADLY NIGHTSHADE (ATROPA BELLADONNA).

THERE are several plants known under the name of Nightshade, which, however, differ much from each other. The WOODY NIGHTSHADE (SOLANUM DULCAMARA), and the GARDEN NIGHTSHADE, or SOLANUM NIGRUM, known by the red and black color of their berries, have been elsewhere noticed (page 647). The vegetable poison now to be described is the DEADLY NIGHTSHADE. The leaves, berries, and root of the plant are poisonous. They owe their noxious effects to the presence of the alkaloid *Atropia.*

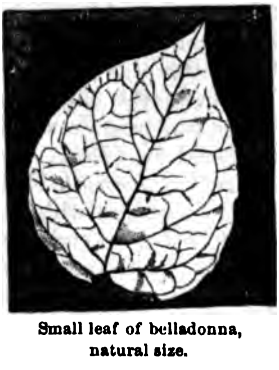
Symptoms.—The symptoms produced by this poison are of a uniform character, and as a summary they may be thus described: Heat and dryness of the mouth and throat, nausea, vomiting, giddiness, indistinct or double vision, delirium, great excitement, convulsions, followed by stupor and lethargy. The pupils are much dilated and the eyes are insensible to light. In two cases which occurred to Mr. Tufnell, the pupils were contracted during sleep, although dilated in the waking state. (*Dub. Med. Press*, Jan. 5, 1853; *Journal de Chimie Médicale*, 1853, p. 695.) Several deaths from the effects of the *berries* occurred in this metropolis in the autumn of 1846. The following case was admitted into Guy's Hospital. A boy, æt. 14, ate, soon after breakfast, about thirty of the *berries* of belladonna, which he had bought in the street. In about three hours it appeared to him as if his face was swollen, his throat became hot and dry, vision impaired, objects appeared double, and they seemed to revolve and run backwards. His hands and face were flushed, and his eyelids swollen; there were occasional flashes of light before his eyes. He tried to eat, but could not swallow on account of the state of his throat. In endeavoring to walk home he stumbled and staggered; and he felt giddy whenever he attempted to raise his head. His parents thought him intoxicated; he was incoherent, frequently counted his money, and did not know the silver from the copper coin. His eyes had a fixed, brilliant, and dazzling gaze; he could neither hear nor speak plainly, and there was great thirst; he caught at imaginary objects in the air, and seemed to have lost all knowledge of distance. His fingers were in constant motion; there was headache, but neither vomiting nor purging. He did not reach the hospital until nine hours had elapsed; and the symptoms were then much the same as those above described. He attempted to get out of bed with a reeling, drunken motion; his speech was thick and indistinct. The pupils were so strongly dilated that there was merely a ring of iris, and the eyes were quite insensible to light. The eyelids did not close when the hand was passed suddenly before them. He had evidently lost the power of vision, although he stared fixedly at objects as if he saw t[illegible]es of common sensation were unaffected. When plac[illegible] could not stand. The pulse was 90, feeble, and compressi[illegible] mouth was in constant motion, as if he were eating something. [illegible]s bladder was full of urine on admission. He continued in this state for two days, being occasionally conscious; when, by a free evacuation of the bowels, some small seeds were passed; these were examined and identified as the seeds of belladonna. The boy gradually recovered, and left the hospital on the sixth day after his admission; the progress of recovery was indicated by the state of the pupils, which had then only acquired their natural size and power of contraction. In three other cases which occurred at the same time, the berries having been baked in a pie, pains in the limbs, drowsiness, insensibility, and convulsions, were among the symptoms.

In two instances of poisoning by the berries related by Dr. Moll, the symptoms bore a strong resemblance to those of delirium tremens, but among them were heat and dryness of the throat, loss of power of swallowing, incoherent speech, double vision, and strange spectral illusions, with occasional fits of wild and ungovernable laughter. On the following morning both patients recovered as if from a dream, but they suffered for some time from languor, thirst, and dryness of the throat; the pupils also continued dilated. (Casper's *Wochenschrift*, January 10, 1846, p. 26.) Two cases of the effects of the berries on children are quoted in the *Edinburgh Medical and Surgical Journal.* (Vol. 29, p. 452.) Among the first symptoms, three hours after the berries were eaten, the children were seized with uncontrollable fits of laughter; catching at objects; incessant incoherent babble, and continued agitation of the body, with fixed staring eyes, and dilated insensible pupils. A man, æt. 34, ate about fifty berries to relieve his thirst. He soon perceived a burning sensation in the throat, and a feeling of stupefaction. He staggered home and went to bed. In the evening he was seized with such violent delirium that it required three men to confine him. His face was livid; his eyes were injected and protruding—the pupils strongly dilated; the carotid arteries pulsated most violently; and there was a full, hard, and frequent pulse, with loss of power to swallow. He was bled, and in about half an hour was able to swallow an emetic; this brought away a violet blue or purple liquid, which is always a well-marked indication of this form of poisoning. Purgative medicines and injections were employed, and the man recovered his consciousness in about twelve hours. (Case by Dr. Rosenberger, Canstatt's *Jahresb.*, 1844, vol. 5, p. 295.) In six other cases, reported in the same journal by Dr. Teschenmacher, the symptoms varied slightly in the different patients. They all experienced double vision, dilatation of the pupils, constriction of the throat, giddiness, and a tendency to sleep. They who had eaten most berries fell into a soporose state, and had violent convulsions of the extremities. In twenty-four hours the whole of the family had recovered.

The *root* of the belladonna, administered in the form of decoction as a clyster, has destroyed life. Four scruples of the root were employed, and the liquid, strained and reduced by evaporation to four ounces, was injected. After a slight stage of excitement, the patient, a woman, æt. 27, fell into a state of complete coma; the countenance appeared swollen, and of a reddish-brown color; the pupils were excessively dilated; the pulse was at first full and hard, then small; death took place in five hours. (Casper's *Wochens.*, Feb. 8, 1845, p. 101.) This case proves that, in poisoning by nightshade, there is in some instances little or no delirium, and that the patient may be at once thrown into a fatal lethargy. A case of recovery, in which a girl, æt. 9, masticated portions of the *root* of belladonna, is reported by Mr. Bullock. (*Med. Gaz.*, vol. 19, p. 265.) In two hours there was sickness, lassitude, and dryness of the throat; in four hours delirium with convulsions, came on; the face was distorted, the eyes were protruded, and the pupils widely dilated. The girl was completely insensible. Under the use of the stomach-pump and emetics she recovered.

The *leaves* of belladonna have occasionally given rise to accidents. A young man swallowed an infusion of two drachms of the leaves. In about an hour he found great difficulty in swallowing, the salivary secretion was suppressed, and objects appeared to be in perpetual motion before him. He became delirious, attempted repeatedly to pass his urine, but could not; and for an hour and a half he was in constant motion, although his gait was unsteady. The muscles of his face, jaws, and limbs were agitated by convulsive twitchings; the pupils were excessively dilated, and there were singular hallucinations. There was neither nausea, vomiting, nor purging. Emetics, injections, and bleeding were resorted to, and the next morning he awoke as if from a dream. The leaves of belladonna are peculiar in shape. The annexed illustration (Fig. 90) is from a photograph of a small leaf of the fresh plant, showing the natural venation of the living leaf. (*Ann. d'Hyg.*, Oct. 1847, p. 413.)

FIG. 90.



Small leaf of belladonna, natural size.

Dr. Garrod has communicated to me the symptoms which one of his patients and himself suffered as a result of taking an infusion of belladonna-leaves which had been ignorantly supplied for ash-leaves. A quarter of an ounce of the leaves was boiled for a few minutes with ten ounces of water. Dr. Garrod took about half a wineglassful of this decoction, equivalent to eleven grains of the dried leaves. He believed, at the time, it was the infusion of ash-leaves, and wished to determine by taste whether it was good. In about half an hour the symptoms commenced by swimming in the head, intense feeling of nervousness, palpitation of the heart, a small and rapid pulse, dryness of the mouth and throat, and perversion of taste, indistinctness of vision, dilatation of the pupils, rapid flow of ideas, weakness of the limbs, and slight difficulty of articulation. His taste was so altered that some brandy given to him had the taste of the infusion. Some of the infusion was put into an eye, and in about a quarter of an hour it dilated the pupil powerfully. In about four hours Dr. Garrod had recovered from these effects; but there was indistinctness of vision with dilated pupils for one or two days, and a generally depressed state of the nervous system, from which even after two years he had not entirely recovered. His patient took half a pint of the infusion, equivalent to about a quarter of an ounce of dried belladonna leaves; the symptoms were similar but more severe, and lasted for a longer time. They were giddiness, difficulty in walking, dryness of the mouth and throat, and perversion of taste, indistinctness of vision, and dilatation of the pupils, bloodshot eyes, difficult articulation, delirium, coma (insensibility), scarlet redness of the skin of the face and neck, followed on the second day by a peeling off of the cuticle. This last symptom, although not common, has been observed in other cases of poisoning by belladonna in large medicinal doses. Some of these symptoms continued more or less for ten hours. The patient suffered from shock to the nervous system for a considerable period after his recovery from the urgent symptoms. One of the effects of the poison was to produce, in both cases, an increased secretion from the kidneys.

The *extract* of belladonna appears to be very uncertain in its operation. It is made from the fresh leaves and young branches of the plants. The medicinal dose is from one-quarter to one grain. In a case which occurred at St. George's Hospital, an ounce of the extract was taken without causing death; but in another instance, a child, æt. 9, nearly lost his life by a dose of thirty grains, administered to him in mistake for extract of taraxacum. Delirium came on in half an hour; this was followed by coma. In addition to other characteristic symptoms, the child suffered from convulsive twitchings of the arms. There was pain in the head, with deranged vision, for ten days after the accident. (*Prov. Med. Jour.*, Feb. 24, 1847, p. 98. See also *Pharmaceutical Journal*, Feb. 1853, p. 404.) Dr. Gray, of New York, has related a case in which a child, between two and three years of age, swallowed from eight to twelve grains of the extract, and after suffering the usual symptoms in a severe form for three hours, recovered. This gentleman describes his own sensations after having taken a large dose of the same preparation. They bear out singularly the truth of the descriptions given by other observers. (See *Med. Gaz.*, vol. 37, p. 255.) Mr. Iliff, Jr., has given an account of the effects produced on *himself* by a dose of nine grains of the extract of belladonna, for which I must refer the reader to the *Lancet.* (Dec. 1, 1849, p. 756. See also for other cases *Med. Times*, Aug. 30, p. 234, and *Ann. d'Hygiène*, 1853, vol. 1, p. 417.) In the last case the members of a family suffered from symptoms of poisoning by the extract, but they all recovered. In the *Medical Gazette* (vol. 42, p. 589) will be found the report of an inquest in a case of alleged poisoning by belladonna, involving many points regarding this poison. The question at issue was whether death had arisen from an overdose of the *extract* or from natural causes. The extract of belladonna is subject to great variation in strength, a fact which may furnish an explanation of certain exceptional cases in which persons are stated to have recovered from large doses of this compound. A case occurred to Mr. Edwards in which a woman, æt. 34, recovered after having swallowed a drachm of the extract by mistake. (*Lancet*, May 24, 1851, p. 568.) Mr. Solly met with an instance in which a man took a scruple by mistake. No symptoms occurred for two hours. He then suffered from dryness of the throat, difficulty of swallowing, fanciful delusions, and rambling, incoherent conversation. The pupils were dilated and insensible to light; the eyes were prominent and had a vacant stare. There was drowsiness, with a feeble and irregular pulse, and a loss of muscular power. Under the use of emetics, the man recovered the next day. (*Lancet*, Feb. 3, 1855, p. 121.)

Two persons swallowed a small spoonful of the extract of belladonna by mistake for that of juniper. There was speedily indistinctness of vision, with tottering gait, delirium, incoherency, hallucinations, and dilatation of the pupils. In one patient there was great cerebral excitement. The apothecary to whom the extract was taken, tasted it, and soon experienced symptoms which led to a suspicion of its real nature. Under treatment, the symptoms of poisoning disappeared in two days; but one of the patients died on the seventh day from disease. The physical and physiological properties of the extract indicated that it was belladonna; but the attempt to procure atropia from it entirely failed. A portion of the concentrated extract given to a dog caused dilatation of the pupil in a quarter of an hour, an index of the rapidity with which the alkaloid atropia is absorbed and diffused through the blood. A woman swallowed on an empty stomach a drachm of the extract of belladonna. She then took some food. After the lapse of three hours, symptoms of poisoning came on suddenly. She lost the power of standing; there was trembling of the limbs with convulsive motions, a nervous laugh, and incoherent speech. The pupils were much dilated, and great lassitude followed this stage of excitement. Under treatment she recovered in twelve hours. (*Ann. d'Hyg.*, Oct. 1847, p. 413.) Orfila has satisfactorily accounted for these anomalies in the power of the extract. Some specimens are quite inert; those only have an energetic action which are prepared by evaporating the fresh juice at a very low temperature. (*Toxicologie*, vol. 2, p. 395.)

Dr. Stevenson met with a case in which a child, between two and three years of age, recovered from a dose of five grains of the extract. (*Guy's Hosp. Rep.*, 1869, p. 268.) In a case of compound poisoning by extract of belladonna and tincture of opium, there was the violent excitement produced by belladonna, but the pupils were strongly contracted as in poisoning by opium. (*Med. Times and Gaz.*, 1870, vol. 1, p. 564.) A case occurred in February, 1865, in which a lady suffered from severe symptoms of poisoning, and nearly lost her life, owing to an injection containing one drachm of the extract of belladonna and one drachm of wine of opium having been administered to her. The pupils in this case were dilated, and the opium did not in any way counteract the effects of the belladonna.

The following case, which occurred in November, 1871, is remarkable for the fact that a woman recovered from a large dose of the *extract*. A nurse gave by mistake to a lady whom she was attending a belladonna liniment containing three drachms of the extract mixed with soap liniment. She had vomited slightly. When seen by Mr. F. Keen, he found her suffering from dryness of the throat, difficulty of swallowing, drowsiness, delirium, the pupils dilated, fixed staring of the eyes, loss of power, and difficulty of speech. Paralysis of the limbs came on with great pain in the back. Emetics with brandy and cayenne pepper were employed with success. The woman recovered, but not until after five weeks from the time of swallowing the liniment.

The *tincture* of belladonna is made from the leaves. The medicinal dose is from five to twenty minims.

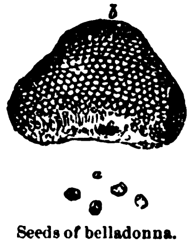
Local Action.—The extract, as it is well known by its effects in dilating the pupil, acts through the *skin*. It is easily absorbed, and must therefore be used with caution. M. Casanova ordered a blister to be applied to the abdomen of a woman, and prescribed a dressing of one part of extract of belladonna to three parts of mercurial ointment. At first nine grains, and, after two hours, thirty grains of the extract were thus employed. The patient was soon attacked with violent delirium, crying out incoherently, and attempting to drive away horrible forms which she fancied she saw flitting around her. The pupil was enormously dilated; there was intense thirst with spasmodic constriction of the throat in drinking. These symptoms did not disappear until after the lapse of forty-eight hours. (*Gaz. Méd.*, Mars 13, 1847, p. 207.) In a case that occurred to Dr. Jenner, symptoms of poisoning arose from the application of a fresh belladonna plaster to a pustular surface produced by the application of an old plaster. Some time afterwards the patient suffered from great dryness of the tongue and throat, which prevented distinct articulation, and was rather increased by his taking water. There was a strong desire to pass the urine, but only a few drops could be passed at a time. There was confusion in the head, and convulsive catchings in the limbs. In about eight hours he had lost the power of standing. He was restless; his hands were in constant motion, as if he were busy in moving light objects. He moved his mouth incessantly, but the sounds thus made were unintelligible. He seemed unconscious of the presence of persons. The pupils were large, and they acted imperfectly under exposure to light. On the removal of the plaster the symptoms ceased, leaving the next day dilatation of the pupils, dimness of vision, and impairment of memory. (*Med. Times and Gaz.*, Nov. 22, 1856, p. 513.)

Appearances.—The appearances observed in several cases of poisoning by the berries which proved fatal in London during the autumn of 1846, were as follows: The vessels of the brain were congested with liquid blood; the stomach and intestines were pale and flaccid; there were some red spots towards the cardiac end. In other fatal cases, of which the appearances have been reported, the brain and its membranes were found distended with thick black blood. Red spots have also been observed around the throat and gullet, and congested patches of a dark purple color on the coats of the stomach. In some instances the mucous membrane has been completely dyed by the juice of the berries. A boy, æt. 5, after having eaten a quantity of the berries of the belladonna, went to bed, was very restless, vomited once, and died in convulsions about fifteen hours after having taken the poison. On inspection, the eyes were half open, with an intense lustre, the pupils dilated, the mouth spasmodically closed, and the sphincter ani relaxed. The cerebral vessels were distended with dark-colored blood, the substance of the brain, cerebellum, and medulla oblongata presented numerous bloody points. In the throat and gullet there were several patches of redness. In the stomach there was some fluid, with three open berries; the mucous membrane was of a reddish-blue color in various parts. (Case by Dr. Rosenberger, Canstatt's *Jahresb.*, 1844, vol. 5, p. 295.)

For another case showing the appearances, see Horn's *Vierteljahrsschrift*, 1866, vol. 2, p. 159. A woman, æt. 66, swallowed a teaspoonful of belladonna liniment, and, after suffering the usual symptoms, died in sixteen hours. On inspection, the lungs were found full of blood, the right side of the heart contained but little black blood, and the left side was firmly contracted. The brain was slightly congested. The stomach and other organs presented nothing unusual. The inspection was made thirty-four hours after death, and the pupils were then dilated. (*Lancet*, 1870, vol. 2, p. 83.)

Analysis.—The indigestible nature of the leaves, fruit, and seeds will commonly lead to their detection in the matters vomited or passed by the bowels, or in the contents of the viscera after death. The *seeds* of belladonna are small, of a somewhat oval shape, and of a dark color. Under a low magnifying power they have a honeycombed surface (Fig. 91). In henbane, the surface of the seeds presents more irregular depressions, resembling those seen on certain corals or madrepores. The seeds of belladonna may be distinguished by the microscope from the seeds of other poisonous plants. In place of the seeds, fragments of the skin of the fruit or berries may be found in the contents of the stomach. The skin gives a purple color to a solution of carbonate of soda, and, in applying spectrum analysis to the liquid, Mr. Sorby found that a portion of skin less than one-tenth of an inch square was sufficient to give a colored solution, in which a characteristic absorption band was produced. It is a narrow dark band, corresponding to that of blood,

FIG. 91.



Seeds of belladonna.
a. Natural size.
b. Magnified 30 diameters.

which is nearer to the red end of the spectrum, and about twice as wide. Mr. Sorby believes this to be characteristic of the solanum purple, found only in the genus solanum.

The coloring matter of the *berry* is of a deep purple hue; it is turned green by alkalies, and red by acids. The leaves (Fig. 90, p. 727) would be known by their botanical characters, and a decoction or infusion of them, by the liquid causing dilatation of the pupil. Dr. Runge found that the urine, blood, or organic liquids containing this poison, applied to the eye of an animal, caused dilatation of the pupil. It takes place equally in poisoning with henbane and stramonium.

ATROPIA.

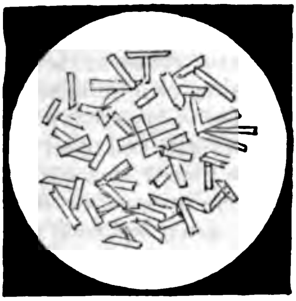
Atropia is the name given to the alkaloidal principal of belladonna; it is a powerful poison. Some consider it to be identical with daturia, the poisonous alkaloid of thornapple, but this is not yet satisfactorily established, either chemically or physiologically. Symptoms of poisoning have been produced by the application of a weak solution of atropia to the eyes. One-eighth of a grain injected beneath the skin, for the relief of sciatica, caused all the symptoms of poisoning with belladonna. One grain used endermically nearly proved fatal to a patient at Guy's Hospital, and in the following case reported by Mr. Leach (*Med. Times and Gaz.*, July 6th, 1865, p. 34), a man who swallowed by mistake a grain of sulphate of atropia, in solution, had a narrow escape of his life. In an hour afterwards, the following symptoms were observed: The pupils were enormously dilated, so that the irides were scarcely visible, and the eyes moved restlessly from side to side. The pulse was very quick, and the patient appeared as if intoxicated. In another hour his hands were cold, the pulse was weak, and there was loss of power in the limbs. He became restless, incoherent, and unconscious of preceding events. There was also delirium. In a later stage there was a morbid sensitiveness to sounds and objects, the tongue was furred, and the skin was dry and hot. The pupils continued dilated for a week, and for several days there was a partial paralysis of the bladder. He recovered in a fortnight. In November, 1850, Mr. Sells, of Guildford, forwarded to me for examination the stomach of a young man who had poisoned himself by taking, as it was supposed, two grains of atropia. He took the dose on going to bed. He was heard to snore heavily during the night, and was found dead about seven o'clock in the morning, lying on his right side, the surface livid, the limbs rigid and contracted, and with a little brown matter issuing from the mouth. The pupils were much dilated. The mucous membrane of the stomach presented a diffused redness, which might have arisen from some brandy which he had swallowed. No trace of the poison could be detected in the stomach or its contents. In the *Association Medical Journal* (Sept. 16th, 1853, p. 818) will be found the report of a case in which all the symptoms of poisoning by belladonna arose from the application of a weak solution of atropia in water to the conjunctiva.

The criminal administration of atropia is a rare event in this country.

A trial for murder by this alkaloid took place at the Manchester Lent Assizes, 1872 (*Reg.* v. *Steele*). The prisoner, who was a nurse in the workhouse, was charged with administering atropia to the senior surgeon, Mr. Harris, and thereby causing his death. The deceased was taken suddenly ill after his breakfast, and he died under the usual symptoms of poisoning with atropia in about twelve hours. The poison was detected in the body by Mr. Calvert, and also in a liquid found in the room—a solution of atropia in spirit. Milk was the vehicle through which it was taken. The milk as sent from the kitchen contained nothing injurious, but that found in deceased's room was tasted by two of the nurses, and they both suffered from poisoning by atropia. The prisoner had access to this room, and it was alleged that she had a strong motive for this criminal act, but there was no direct proof to show that she put the poison into the milk, and she was acquitted.

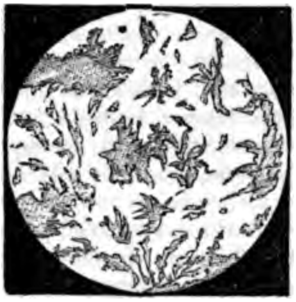
Analysis.—Atropia is a white crystalline substance, requiring 500 parts of water to dissolve it, but easily dissolved by alcohol, ether, and diluted acids. It may be crystallized from its alcoholic solution, but with some difficulty. The annexed illustration (Fig. 92) shows the crystalline form of pure atropia, given to me by the late Mr. Morson, and Fig. 93 represents the irregular crystalline forms of the sulphate of atropia, as it is deposited from an alcoholic solution. Ammonia added to the solution of sulphate of atropia does not separate the alkaloid in distinct crystals. In this respect it differs from morphia and strychnia. When atropia is heated on platinum it melts, darkens in color, and burns with a yellowish smoky flame. Sulphuric and hydrochloric acids dissolve it without coloring it. Nitric and iodic acids produce with it an ochreous color. Sulphomolybdic acid produces no change. Tannic acid precipitates the alkaloid from its solutions; but the most effectual precipitant is the chloriodide of potassium and mercury, which throws down a dense white precipitate even in very diluted solutions. Atropia is also precipitated by chloride of gold, but unlike strychnia, it is not precipitated by sulphocyanide of potassium or chromate of potash. According to Winckler, atropia is most completely

FIG. 92.



Crystals of pure atropia, magnified 70 diameters.

FIG. 93.



Imperfect crystals of sulphate of atropia, magnified 80 diameters.

precipitated from all its solutions by the chloriodide of potassium and mercury (p. 527, *ante*). By the use of this precipitant he was able to determine the proportion of atropia contained in the powder of the dry leaves and root. In the leaves the alkaloid varied from 0.41 to 0.49 per cent., and in the root it amounted to 0.48 per cent. (*Pharm Jour.*, June, 1872, p. 1029.) According to Mr. Luxton, 1000 grains of the leaves yield only five grains of atropia. The pharmaceutical preparations of this alkaloid are, 1, a solution, *Liquor atropiæ*, in rectified spirit and water in the proportion of four grains of atropia to one fluid ounce. 2. The *Liquor atropiæ sulphatis*, in which the sulphate is in a similar proportion; and, 3. The ointment, which contains eight grains to the ounce.

Organic Liquids.—Atropia may be separated from organic liquids by a process similar to that which has been elsewhere described for strychnia. (See *ante*, p. 688.) There are no absolute or certain chemical tests for this alkaloid when contained in an organic liquid. The only test usually employed is of a physiological nature, namely, the effect produced on the pupil of the eye by small quantities of liquid, or extract, containing traces of atropia. The pupil is largely dilated by the salts of this alkaloid, and the eye loses its sensibility to light. The introduction of any organic extract, containing atropia, into a wound in the cellular membrane of an animal, also causes dilatation of the pupil. Daturia, hyoscyamia, and digitalin applied to the eye, also produce dilatation of the pupil. Poisonous mushrooms and other noxious organic matters have a similar effect, so that there is nothing very conclusive in this result, unless there is also strong evidence from *symptoms* that belladonna has been actually taken or administered.

At the Exeter Autumn Assizes for 1865 (*Reg.* v. *Sprague*), a medical man was charged with attempting to poison his wife and other persons with atropia, which it was alleged had been placed in a rabbit pie. The evidence failed to show at the trial that the prisoner, or any other person, could have mixed poison with the pie, much less such a poison as this, which, in the dose of one or two grains, either destroys life or produces serious illness continuing for some time. The symptoms, as described, resembled those caused by noxious food, and differed in many respects from those of poisoning with atropia. The only fact on which this chemical theory seemed to rest was, that the pupils of those who ate of the pie, and were taken ill afterwards, were dilated, and a portion of the extract of the scrapings of the pie-dish is said to have caused a dilatation of the pupil of the analyst. (See *Med. Times and Gaz.*, August 12, 1865, p. 168; also *Chemical News*, August 11, 1865, p. 72.) It is stated that the supposed poison was separated from the baked leg of a rabbit by soaking it in dilute hydrochloric acid; but, according to those who have examined the properties of atropia, this alkaloid melts at 194°, is entirely volatile under 300°, and is then in great part decomposed. This is below an ordinary cooking temperature. (*Chemie der Organischen Alkalien*, Schwartzkopf, p. 317.) The whole of the scientific theory rested upon the dilatation of the pupils, and this, although presumptive, is not positive evidence of atropia having been administered.

CHAPTER LXXIV.

POISONING WITH LOBELIA OR INDIAN TOBACCO—DATURA STRAMONIUM OR THORN-APPLE—SYMPTOMS AND APPEARANCES—HOCUSSING—DATURIA: ITS ACTION AND CHEMICAL PROPERTIES.

INDIAN TOBACCO (LOBELIA INFLATA).

THE powdered leaves of Indian tobacco contain an acrid principle which is capable of producing poisonous effects on the brain and spinal marrow, attended with irritation of the stomach and bowels. Wibmer relates that in one instance it produced at first violent vomiting in the person for whom it was prescribed; but the medicine was repeated until it was no longer ejected from the stomach. The patient suffered severe pain, and speedily died, stupor and convulsions having preceded death. The powdered leaves and seeds have been much employed by quacks in the United States, and accidents have occasionally arisen from the substance having been taken in excessive doses.

When administered in doses of from ten to twenty grains, lobelia operates as an emetic; but in larger quantity it acts deleteriously. It would also appear that even ordinary medicinal doses affect some persons with great severity. There is an erroneous notion that this is a useful medicine and not a poison, although, like arsenic and opium, it may be either, according to the mode in which it is employed.

In one case a man lost his life by swallowing *one drachm* of the powdered *leaves*, prescribed by a quack. This person was seen by a medical practitioner soon after he had taken the poison. He was evidently suffering great pain, but he was quite unconscious; the pulse was small, and the pupils were strongly contracted and insensible to light. He had vomited the greater part of the poison. He suffered from spasmodic twitchings of the face, sank into a state of complete insensibility, and died in about thirty-six hours. On inspection, some fluid was found in the stomach, but none of the powder. The mucous membrane was intensely inflamed, and the vessels of the brain were strongly congested. (*Pharm. Times*, May 1, 1847, p. 182.) The *seeds* of lobelia are equally poisonous. In the *Medical Times and Gazette*, November 26, 1853, p. 568, two cases are reported in which the seeds proved fatal. In one the mucous membrane of the stomach was highly inflamed. Another case is referred to in the same journal, March 12, 1853, p. 270.

There have been many inquests and trials for manslaughter in this country as the result of the improper administration of the powdered *leaves* of the *lobelia inflata* by ignorant quacks, calling themselves medical botanists and dealers in vegetable medicines. The medical evidence given on these trials has proved that in large doses, lobelia is a

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mercury (p. 527, *ante*). By the use of this precipi
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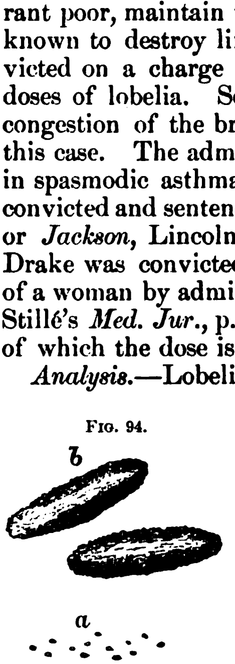
Analysis.—Lobelia is seen in the form of a greenish-colored powder (fragments of leaves). This powder acquires a reddish-brown color from strong nitric acid, and is blackened by concentrated sulphuric acid. Iodine-water has no effect upon the infusion. The proto- and persulphate of iron produce with it a dark green color, the persulphate very rapidly. The leaves and seeds contain a resinoid substance called *lobelin*, which has the smell and taste of the plant. It acts as a powerful emetic in doses of from one-half to one grain. The leaves of lobelia are generally seen in fragments, which do not readily admit of identification by the microscope. The *seeds* are very small, of a lengthened oval shape, reticulated on the surface with projecting hairs or fibres, and of a light-brown color (Fig. 94). The discovery of them among the fragments of leaves would furnish a sufficient proof of the presence of lobelia.

Fig. 94.



Seeds of lobelia.
a. Natural size.
b. Magnified 70 diameters.

THORNAPPLE (DATURA STRAMONIUM).

All parts of this plant are poisonous; but the seeds and fruit are considered to be the most noxious. From a case published by Dr. Zechmeister, it would appear that even the *vapor* of the full-blown *flowers* may give rise to symptoms of poisoning. The case was that of a boy who breathed the vapor for some time in a close apartment. (*Œsterreich Med. Woch.*, July 19, 1845.)

Symptoms.—The usual effects produced by this poison will be understood from the following cases. A woman, æt. 36, took two teacupfuls of infusion of stramonium *leaves*, by mistake for senna tea. In about ten minutes she was seized with giddiness, dimness of sight, and fainting. In two hours she was quite insensible; the pupils were fixed and

dilated, all the muscles of the body convulsed, the countenance flushed, and the pulse was full and slow. The stomach-pump was applied, and in the course of a few hours she recovered; suffering, however, from indistinctness of vision and vertigo. (*Med. Gaz.*, vol. 8, p. 605.) In the *Lancet* (April 26, 1845, p. 471), a case quoted from the *Boston Journal*, in which three women swallowed an infusion of stramonium *leaves* for horehound. They were found lying in bed, stupid, unable to articulate, with a peculiar wildness of countenance and flushed face; the pupils were dilated and insensible, the conjunctivæ highly injected, lips and tongue parched, no vomiting, breathing at times stertorous and labored, hands cold, with a trembling and slightly convulsive movement, great rigidity of the muscles of the neck and back, and occasionally active efforts at utterance. Stimulants were administered with benefit in two cases; the third proved fatal.

The *seeds* of this plant have been known to produce furious delirium; and a case is mentioned by Sauvages of an old man of sixty, who, after taking the poison, became intoxicated, maniacal, and lost the power of speech. He remained in a lethargic state for five hours. Several fatal cases are reported, one of which terminated in six hours. Dr. Thomson relates the case of a child, aged two years, who swallowed sixteen grains of the seeds. Maniacal delirium supervened; the symptoms resembled those of hydrophobia, and death took place in twenty-four hours. A case which occurred to Dr. Schlesier ended more fortunately. A boy, æt. 4, mistaking the fruit of the thornapple for the heads of poppies, ate a quantity of them. Dr. Schlesier saw him soon afterwards; his face was flushed, his eyes were glistening and in constant motion, the pupils much dilated, and the countenance was that of an intoxicated person. He sat up in bed quite unconscious, but continually babbling and occasionally starting up suddenly, his hands apparently directed at imaginary objects in the air. His pulse was very slow; there was no fever, but intense thirst and violent perspiration from incessant motion. Emetics and injections were administered, which had the effect of bringing away a large quantity of stramonium seeds; the boy fell into a sound sleep, and recovered on the following day. (Canstatt's *Jahresb.*, 1844, p. 297.) Mr. Sobo met with the case of a child, æt. 5, who ate more than a drachm of the seeds slightly roasted. In about an hour he appeared much excited and delirious, pulse 120, face flushed, eyes of a dazzling lustre, and pupils dilated; there were convulsive motions of the limbs and neck, with thick, frothy saliva about the mouth. Emetics were given, some stramonium seeds were ejected, and more were brought away in the evacuations by a full dose of castor oil. In three days the boy had perfectly recovered. (*Med. Times*, Oct. 9, 1847, p. 650. For other cases, see *Prov. Journal*, Dec. 24, 1851, p. 699; and *Lancet*, May 31, 1851, p. 599.) Paralysis and delirium have been witnessed among the symptoms, which on the
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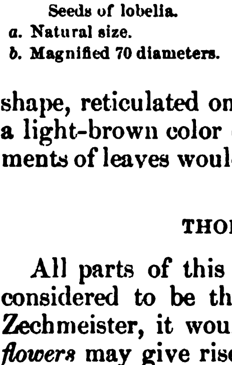

Seeds of lobelia.
a. Natural size.
b. Magnified 70 diameters.

THORNAPPLE (DATURA STRAMONIUM).

All parts of this plant are poisonous; but the seeds and fruit are considered to be the most noxious. From a case published by Dr. Zechmeister, it would appear that even the *vapor* of the full-blown *flowers* may give rise to symptoms of poisoning. The case was that of a boy who breathed the vapor for some time in a close apartment. (*Œsterreich Med. Woch.*, July 19, 1845.)

Symptoms.—The usual effects produced by this poison will be understood from the following cases. A woman, æt. 36, took two teacupfuls of infusion of stramonium *leaves*, by mistake for senna tea. In about ten minutes she was seized with giddiness, dimness of sight, and fainting. In two hours she was quite insensible; the pupils were fixed and

dilated, all the muscles of the body convulsed, the countenance flushed, and the pulse was full and slow. The stomach-pump was applied, and in the course of a few hours she recovered; suffering, however, from indistinctness of vision and vertigo. (*Med. Gaz.*, vol. 8, p. 605.) In the *Lancet* (April 26, 1845, p. 471), a case quoted from the *Boston Journal*, in which three women swallowed an infusion of stramonium *leaves* for horehound. They were found lying in bed, stupid, unable to articulate, with a peculiar wildness of countenance and flushed face; the pupils were dilated and insensible, the conjunctivæ highly injected, lips and tongue parched, no vomiting, breathing at times stertorous and labored, hands cold, with a trembling and slightly convulsive movement, great rigidity of the muscles of the neck and back, and occasionally active efforts at utterance. Stimulants were administered with benefit in two cases; the third proved fatal.

The *seeds* of this plant have been known to produce furious delirium; and a case is mentioned by Sauvages of an old man of sixty, who, after taking the poison, became intoxicated, maniacal, and lost the power of speech. He remained in a lethargic state for five hours. Several fatal cases are reported, one of which terminated in six hours. Dr. Thomson relates the case of a child, aged two years, who swallowed sixteen grains of the seeds. Maniacal delirium supervened; the symptoms resembled those of hydrophobia, and death took place in twenty-four hours. A case which occurred to Dr. Schlesier ended more fortunately. A boy, æt. 4, mistaking the fruit of the thornapple for the heads of poppies, ate a quantity of them. Dr. Schlesier saw him soon afterwards; his face was flushed, his eyes were glistening and in constant motion, the pupils much dilated, and the countenance was that of an intoxicated person. He sat up in bed quite unconscious, but continually babbling and occasionally starting up suddenly, his hands apparently directed at imaginary objects in the air. His pulse was very slow; there was no fever, but intense thirst and violent perspiration from incessant motion. Emetics and injections were administered, which had the effect of bringing away a large quantity of stramonium seeds; the boy fell into a sound sleep, and recovered on the following day. (Canstatt's *Jahresb.*, 1844, p. 297.) Mr. Sobo met with the case of a child, æt. 5, who ate more than a drachm of the seeds slightly roasted. In about an hour he appeared much excited and delirious, pulse 120, face flushed, eyes of a dazzling lustre, and pupils dilated; there were convulsive motions of the limbs and neck, with thick, frothy saliva about the mouth. Emetics were given, some stramonium seeds were ejected, and more were brought away in the evacuations by a full dose of castor oil. In three days the boy had perfectly recovered. (*Med. Times*, Oct. 9, 1847, p. 650. For other cases, see *Prov. Journal*, Dec. 24, 1851, p. 699; and *Lancet*, May 31, 1851, p. 599.) Paralysis and delirium have been witnessed among the symptoms, which on the
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Analysis.—Lobelia is seen in the form of a greenish-colored powder (fragments of leaves). This powder acquires a reddish-brown color from strong nitric acid, and is blackened by concentrated sulphuric acid. Iodine-water has no effect upon the infusion. The proto- and persulphate of iron produce with it a dark green color, the persulphate very rapidly. The leaves and seeds contain a resinoid substance called *lobelin*, which has the smell and taste of the plant. It acts as a powerful emetic in doses of from one-half to one grain. The leaves of lobelia are generally seen in fragments, which do not readily admit of identification by the microscope. The *seeds* are very small, of a lengthened oval shape, reticulated on the surface with projecting hairs or fibres, and of a light-brown color (Fig. 94). The discovery of them among the fragments of leaves would furnish a sufficient proof of the presence of lobelia.

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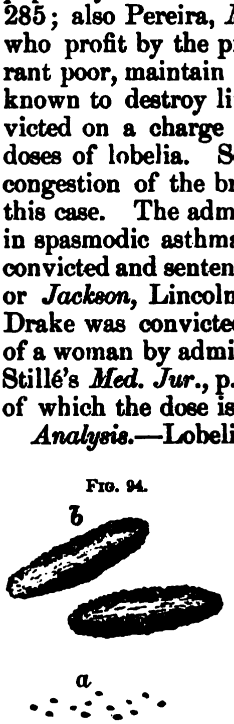

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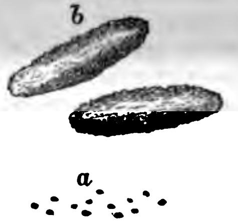

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A boy, æt. 5, ate some stramonium seeds with a portion of the plant.

Soon afterwards it was observed that his face was flushed, and that he staggered as if intoxicated. He vomited, and threw up about thirty seeds. His skin was hot and red, the countenance had a wild and staring expression, the pupils were nearly fully dilated, and insensible to light. The child was restless, in a state of raging delirium, and biting with fury at those who attempted to restrain him. He was unable to stand, and in a state resembling St. Vitus's dance. The pulse could not be counted. The breathing was hurried and gasping. He was incessantly talking, but without articulating distinctly, and he appeared to be driving away from him imaginary objects. Emetics produced the vomiting of more seeds, and in an hour he began to articulate. He slept restlessly for two hours. Some seeds were passed in the evacuations from the bowels. In four hours the symptoms had abated, and the boy gradually improved. The pupils did not recover their natural state until after three days. (*New York Journal of Medicine*, 1856, and *Brit. and For. Med. Rev.*, 1857, vol. 19, p. 497.)

In the *American Journal of Medical Sciences*, April, 1864, p. 552, Dr. Turner describes five cases of poisoning by the seeds, in children under ten years of age. They had eaten them in the scarcely ripe state, when they are not very bitter. In one hour and a half two of the children were found to be fully under the influence of the poison. They were lying on their backs, eyes bright, pupils widely dilated and insensible to light, conjunctivæ injected, face deeply suffused, and of a dark crimson color; difficulty of breathing, inability to articulate, and in a state of complete insensibility, broken occasionally by a paroxysm, during which they would utter some indistinct sounds and throw their hands about, as if trying to ward off some threatening evil. They then fell into a comatose state, but were easily roused into a state of violent excitement; they grasped at imaginary objects; there was picking of the bed-clothes, with paroxysms of excessive laughter. They had no proper control over their limbs, walked with a staggering gait, and fell to the ground as if intoxicated or in a state of complete exhaustion. They recovered under treatment in about twenty-four hours. (See also other cases by Dr. Lee, in the same journal, Jan. 7, 1862, p. 54.)

Death may take place although the whole of the seeds are ejected. This happened in a case reported by Mr. Duffin, that of his own child, æt. 2, who swallowed about one hundred seeds of stramonium, weighing sixteen grains. The usual symptoms were manifested in an hour, and the child died in twenty-four hours, although twenty seeds had been ejected by vomiting and eighty by purging. (*Med. Gaz.*, vol. 15, p. 194.) Sufficient daturia to destroy life had been absorbed from the entire seeds and carried into the blood. In a case which became the subject of a trial at Osnabrück, a woman administered to her mother a decoction of the bruised *seeds* of the thornapple, of which it was supposed there were about 125. She very soon became delirious, threw her arms about, and spoke incoherently; she died in seven hours. (Henke, *Zeitschrift der S. A.*, 1837, i. H.) The seeds retain their properties notwithstanding exposure to heat; thus the smoking of stramonium seeds is attended with danger. In the return of the Registrar-General for April, 1856, there is the record of one death from this cause.

One of the methods of poisoning adopted by the Hindoos, not so much with the intention of destroying life as of facilitating the perpetration of robbery, consists in administering to persons either the powdered seeds in cakes, or a strong decoction of them in curry or some other highly flavored article of food. Drowsiness, delirium, and insensibility soon follow, and sometimes death is the result, but no suspicion of the real cause appears to be excited.

Dr. Brown, of Lahore, states that out of ninety-two of these cases of poisoning no fewer than twenty-one proved fatal, but it is probable that many which result in death are never known, while those who survive would naturally complain of any injury that might have been done to them while insensible. He observes that the drug has a bitter taste, which it generally imparts to the food with which it is mixed, and which is sometimes recognized when it is eaten. The symptoms usually occur in about ten minutes after the poison has been taken, although they may be delayed from half an hour to an hour. There is at first dryness in the throat, great thirst, attended with a feeling of faintness, headache, and giddiness, and the person has difficulty in walking straight; he staggers, and appears as if intoxicated, while at the same time he is very restless. The pupils of the eyes, if examined, are found to be dilated, and he will sometimes complain of indistinctness of vision, or drowsiness, and he almost always falls asleep. The sleep may either increase to complete insensibility, with dilated pupils, a flushed face, and muttering delirium, or the patient may awake and then become delirious. The delirium is characterized by great restlessness, the person affected frequently moving about, and there is a tendency to go naked and to pick at various objects. The pulse is generally slow, the pupils are dilated, and there is great thirst. After a time the patient becomes again insensible, and is greatly exhausted; sometimes convulsions occur, with low muttering delirium, and at length he dies. If, as it more frequently happens, he recovers, the insensibility persists for a day or more, and the patient remains occasionally in an idiotic or lethargic state, able to speak, but not to understand for some time longer, and he has in general no recollection of what has occurred after the poisonous meal. Sometimes vomiting is an early symptom, and the seeds of datura may be found in the vomited matter. Vomiting is not, however, a common symptom. (*Description of Poisons in the Punjab*, 1863, p. 57.)

Dr. Chevers has given a very complete account of the Hindoo system of poisoning by *dhatoora*. (*Med. Jur. for India*, 1856, pp. 121, 549, 591.) It appears that the *Datura fastuosa* and *alba* are the principal sources of the poison in India. The Thugs employed this poison with the object of rendering their intended victims helpless. As it is administered by skilled professional poisoners in India, it causes a profound lethargy resembling coma, with dilated pupils. The person is rendered completely powerless. These symptoms may continue for two days, and yet recovery take place. He also states that the cases rarely prove fatal. Out of fifty-one instances of poisoning by dhatoora, at the Bombay Hospital in one year, recorded by Dr. Giraud, one only was fatal, but four presented very alarming symptoms. Dr. Chevers noticed among them the early occurrence of insensibility. A man drank two mouthfuls of a poisoned liquid, complained of a bitter taste, and fell down insensible within forty yards of the spot where he had drunk the liquid, and did not recover his senses until the third day after. (*Op. cit.*, p. 137.) In these cases, probably the seeds are given in a large dose, either in solution or in very fine powder.

The first stage of poisoning is commonly marked by delirium; the patient is restless, and wanders about as if in search of something, and he is evidently under some strange hallucinations. Owing to giddiness or great muscular weakness, he is soon unable to walk or even to stand; he talks incoherently, laughs wildly, moves about as if to avoid spectra, and picks or catches incessantly at real or imaginary objects. He appears as if drawing out imaginary threads from the ends of his fingers, and his antics are of the most varied and ludicrous kind. The pupils are invariably dilated, and the spectra are illusions depending on disordered vision. Distant objects appear near to him, and near objects as if highly magnified; he will attempt to grasp a distant object as if it were close at hand, and will start back on a person approaching, as if he thought the person was quite near him. In the second stage of poisoning, there is either great drowsiness or complete stupor, sometimes passing into utter insensibility, with stertorous breathing. The third stage of final delirium is similar to the first. (*Op. cit.*, p. 593.)

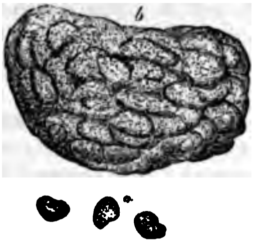
I am indebted to Dr. Irving, of Allahabad, for some additional information respecting the employment of datura by the professional poisoners of India. (*Cases of Food-Poisoning, etc.*, 1864.) According to the observations made by this gentleman, the effects are generally produced within a quarter of an hour after the poison has been taken, and those who have taken the poisoned food have had little or no recollection of anything that occurred afterwards. An extract of datura is probably used as one of the methods of "hocussing" persons by thieves in this country. The dilatation of the pupil, with the peculiar train of symptoms above described, would distinguish this state from ordinary intoxication. The bitter taste of the poisoned liquid might excite suspicion; but, if the person is already partially intoxicated, he may be incapable of making any observation of this kind.

The *local application* of the bruised leaves, seeds, or fruit to an abraded portion of skin may give rise to all the effects of poisoning.

The *extract* of stramonium possesses the properties of the seeds, producing, in an overdose, dryness of the throat, intoxication, and delirium. Dr. Traill met with two cases of poisoning by this substance, in one of which eighteen grains of the extract were taken by mistake for extract of sarsaparilla. (*Outlines*, p. 141.) The medicinal dose of the extract is from a quarter to half a grain; of the powdered seeds, half a grain; and of the leaves, one grain. The seeds and leaves are seldom prescribed medicinally, but are used by smokers. A *tincture* is made from the seeds, the medicinal dose of which is from ten to thirty minims.

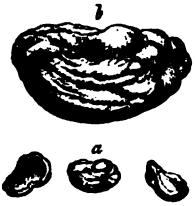
Dr. Irving describes the appearances met with in the body of one of the professional Indian poisoners, Bassawur Singh, who, in order to lull suspicion, partook of the poisoned food himself. His intended victims became insensible; he robbed them and left them to their fate. After a time they recovered their senses, and gave information at the police-station. The poisoner was found under a tree, about a mile from the place, quite insensible. Remedies were unsuccessfully used, and he died shortly after being apprehended. On his person was found all the stolen property, besides a quantity of datura seeds. The following were the post-mortem appearances: The pupils were widely dilated; the body was covered with dust, as if it had been rolled on the ground. The fingers of both hands were firmly clenched. There was great venous congestion of the brain and membranes; slight effusion of bloody serum under the membranes, chiefly on the right hemisphere. About an ounce of dark fluid blood was found at the base of the skull. The bloody points on a section of the brain were numerous. The ventricles contained a considerable quantity of serum. The choroid plexus was unusually full of blood. In the stomach there was a quantity of food, partly digested, in which were found seeds of datura as well as seeds of the *Solanum melongena*, which in form they somewhat resembled. (*Cases of Food-Poisoning*, etc., 1864. *Indian Annals of Medical Science*, No. 17.) Congestion of the lungs has been found as well as great congestion of the mucous membrane of the stomach and intestines, with red patches of extravasated blood in the large intestines.

FIG. 95.



Seeds of datura stramonium.
a. Natural size.
b. Magnified 30 diameters.

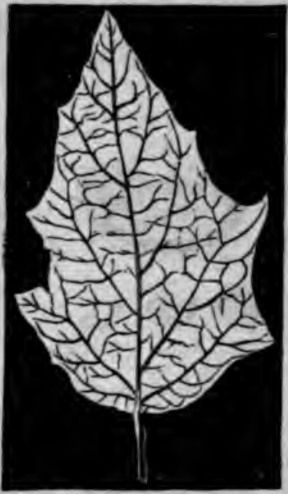
FIG. 96.



Seeds of datura alba (India).
a. Natural size.
b. Slightly magnified by a lens.

Appearances.—In a well-marked case of poisoning by stramonium seeds, in which death took place in less than eight hours, Mr. Allan found the following appearances: Great congestion of the vessels of the brain and its membranes, the brain firm and highly injected, choroid plexus turgid, ventricles containing serum, substance of the lungs congested, the heart flaccid. The stomach contained about four ounces of digested food mixed with eighty-nine seeds of stramonium. There were two patches of extravasation in the mucous coat—one on the larger curvature, and the other near the pylorus. Many seeds and fragments were also found in the intestines. (*Lancet*, Sept. 18, 1847, p. 298.) In the Osnabrück case (p. 738) there were marks of diffused inflammation about the cardia. In Mr. Duffin's case there was nothing remarkable in the condition of the brain or its membranes: no seeds were found in the intestinal canal.

FIG. 97.



Small leaf of stramonium, from a photograph: natural size.

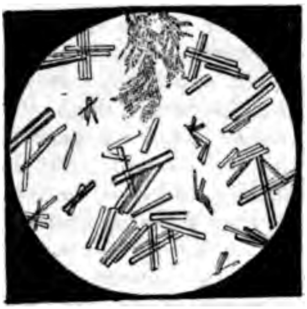
Analysis.—The *seeds* of stramonium, from which accidents have most frequently occurred, are flattened, kidney-shaped, but half oval, rough, and of a dark-brown or black appearance (Fig. 95, p. 741.) The seeds are liable to be mistaken for those of capsicum. Dr. Brown thus describes the difference: "The datura seeds present dots on their exterior, which on a microscopical examination are seen to be composed of convoluted ridges surrounding spaces. On the capsicum seeds these convoluted ridges run nearly parallel to each other, and are joined at right angles by shorter ridges, so that most of the spaces are of an oblong form, and are as lines curving round the seed; but in datura, the ridges are more convoluted and irregular, joining at acute angles and circumscribing irregular spaces." (*Poisons of the Punjab*, 1863, p. 67.) Of the dry datura stramonium, there are about eight seeds to a grain. They are of an oblong kidney-shape, and of a dark-brown or black color. The seeds of the datura fastuosa, received from Dr. Brown, of Lahore, are so similar in size and general appearance that a separate illustration of them is quite unnecessary. The seeds of datura alba, also received from Dr. Brown, are larger, flatter, and much lighter-colored, but have similar microscopical characters (Fig. 96, p. 741). These are the seeds which are chiefly used by the Thugs and the poisoning robbers of India.

The *leaves* of the common datura stramonium are well characterized by their peculiar shape. In the above illustration (Fig. 97) is represented a small leaf of the datura stramonium from a young plant. In the full-grown plant the leaves retain the same characters, but are much larger. It has been engraved from a photographic impression of a fresh leaf of the plant, and shows by dark lines the venation of the leaf.

DATURIA.

The poisonous properties of thornapple are owing to the presence of an alkaloid, *daturia*, which forms about one per cent. of the dried vegetable. For a comparison of its properties with those of atropia and hyoscyamia, which it resembles, see Bouchardat, *Ann. de Thérapeutique*, 1864, p. 24. This alkaloid crystallizes in long colorless prisms or needles (Fig. 98), it has a bitter taste, somewhat acrid, and slightly resembling that of tobacco. It is poisonous. The eighth of a grain killed a sparrow in three hours. When placed on the eye, or introduced into the cellular membrane of an animal, it is observed, like atropia, to cause a dilatation of the pupil, which may last for some days. When heated in a tube it is decomposed, and ammonia is evolved, as with other alkaloids. It is soluble in boiling water, and the solution has an alkaline reaction. It is precipitated by tannic acid and by the chloriodide of potassium and mercury. Nitric, iodic, and hydrochloric acids dissolve it, without producing any change of color. Sulphuric acid produces a pale red color with the crystals, which becomes paler when the acid mixture is diluted with water. Sulphomolybdic acid produces no immediate change.

FIG. 98.



Crystals of daturia, magnified 30 diameters.

The absorption of this poisonous alkaloid is doubtless the cause of the symptoms. Mr. Allan, in the case above related, states that he obtained from six ounces of urine, taken from the bladder of the deceased, crystals of daturia; but they appear to have been of an entirely different form, *i. e.*, pentahedral or polyhedral plates, instead of quadrangular prisms. They resembled daturia only in causing dilatation of the pupil when dissolved in water and the solution was dropped into the eye. Their form appears to have been that assigned to cystin by microscopical observers. (Bird's *Urinary Deposits*, p. 146.)

CHAPTER LXXV.

LABURNUM—CYTISIN—ACTION OF THE BARK AND SEEDS—YEW LEAVES AND BERRIES—PRIVET—HOLLY—GUELDER ROSE—QUINOIDINE—CURARA AND CURARINA.

LABURNUM (CYTISUS LABURNUM).

Symptoms and Effects.—The bark and seeds of the common LABURNUM contain an active poison called *Cytisin*. A case of poisoning by the *bark*, which was the subject of a trial at Inverness, has been reported by Sir R. Christison. (*Ed. Med. and S. J.*, Oct. 1843.) A youth, with the intention of merely producing vomiting in one of his fellow-servants, put some dry laburnum-bark into the broth which was ng prepared for their dinner. The cook, who remarked a "strong uliar taste" in the broth, soon became very ill, and in five minutes ith violent vomiting. The account of the symptoms is e cause of them was not even suspected until six months he vomiting continued thirty-six hours; was accompa- ng, pain in the abdomen, especially in the stomach, and , with severe purging. These symptoms continued, r a period of eight months; and the woman fell off in u s th. At this period she was seen by a physician, who n c on by the law authorities to investigate the case. She n s ing from gastro-intestinal irritation, vomiting after food, t domen, increased by pressure, purging, tenesmus, and ons, with other serious symptoms. The medical opin- in a hig dangerous state. The woman eventu until th lowing April. There was no doubt, from the inves n made by Dr. Ross and Sir R. Christison, that her protracted illness was really due to the effects of the laburnum-bark.

Some experiments were then made by these gentlemen on the action of the poison on animals. A teaspoonful of the powder of dry laburnum bark was administered to a cat. Soon afterwards it writhed, apparently in great pain; in a short time it vomited violently, and, although languid and dejected for the rest of the day, it quickly recovered. Sixty-nine grains of the same powder were given to a dog. In ten minutes it whined and moaned, vomited violently, and soon got well. On a second occasion, twenty grains were found to act as a powerful emetic upon the animal. An ounce of the infusion of laburnum bark, containing the active matter of sixty-two grains, was introduced by a catheter into the stomach of a full-grown rabbit. In two minutes the animal looked quickly from one side to the other, twitched back its head twice or thrice, and instantly fell upon its side in violent tetanic convulsions, with alternating emprosthotonos and opisthotonos, so energetic, that its body bounded with great force upon the side, up and down the room. Suddenly, in half a minute more, all movement ceased, respiration was at an end, the whole of the muscles became quite flaccid, no sign of sensation could be elicited, and the animal died within *two minutes and a half* after the poison was injected into the stomach. The body was opened in two minutes more, and the heart was found gorged with blood, but contracting with some force. The stomach was filled with green pulp, soaked with the infusion. No morbid appearance was visible anywhere. In repeating this experiment, one rabbit died in half an hour, another in three-quarters of an hour, after small doses of the infusion were injected into the stomach; and a third rabbit speedily died after eating greens merely impregnated with the infusion. (*Ed. Med. and Surg. Journal*, 1843, vol. 60, p. 303.) In all these instances convulsions were the leading symptoms produced. The same effects are popularly ascribed to the leaves, young pods, and seeds of the tree; but no experiments have been performed with them. The facts here detailed show that laburnum bark is an energetic poison.

The effects of this bark as a poison were observed in a case which occurred to Mr. Tinley, of Whitby. A girl, æt. 18, idly and unthinkingly put a small portion of a laburnum branch into her mouth, carrying it for some hours, and chewing it. It was described as of the thickness of the little finger, and two or three inches long. There were some yellow flowers with it, but she was not aware that she had swallowed any. In about half an hour she felt unwell, but she was not seen by Mr. Tinley until the day following. The symptoms then were great pain in the stomach, nausea and retching, but no vomiting; pulse 100, tongue white, great thirst, anxiety and pallor of countenance, dilated pupils, sense of fainting, even while lying down, and great exhaustion. There was no purging. Under treatment these symptoms disappeared, and the girl recovered in about a fortnight. (*Lancet*, 1870, p. 182.)

In reference to poisoning by the *seeds* there are but few instances recorded. Dr. Traill has described two cases, and Mr. Rake, a former pupil, has communicated to me a case of poisoning by the pods and seeds of laburnum which occurred in September, 1851. Two children, the one aged two, and the other three years, had been playing together, and on returning home they appeared unwell, and soon afterwards vomited. They had been seen with laburnum pods in their hands, and some seeds with portions of the pods were mixed with the vomited matter. Both children were pale and exhausted, with a slow and somewhat feeble pulse. The pupils were natural. An emetic was given, but no more seeds were ejected; the pulse increased in volume and frequency, and the next day the children had recovered their usual health. In October, 1856, twelve children, at Otley, in Yorkshire, were attacked with rigidity of the limbs and other symptoms of poisoning in consequence of having swallowed these seeds. They recovered under the use of emetics. (*Lancet*, Nov. 1, 1856, p. 497.)

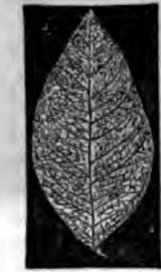
In September, 1862, two boys swallowed a quantity of laburnum seeds in a cake. In about three-quarters of an hour one was seized with vomiting and purging, pulse weak and frequent, severe rigors, muscular twitchings in the face and neck, and great epigastric pain. The pupils were dilated, but there was no headache. Many seeds were vomited. There was a great disposition to sleep, and coldness of the skin. Under treatment they recovered. (*Pharm. Journal*, Oct. 1862, p. 185.) In September, 1863, a girl, æt. 9, died at Worcester in consequence of having eaten a few of the seeds. A boy, æt. 4, ate about ten of the seeds. In half an hour he began to vomit, the vomited matter consisting of food and thick mucus. He afterwards became drowsy, and was seized with convulsions, shaking violently and drawing up his limbs at intervals. Although drowsy, he was easily roused, but soon dozed off again. Both pupils were largely dilated, pulse small, 85, surface, especially of the limbs, cold. He fell into a calm sleep, and the next day he was well. (*Lancet*, 1871, vol. 2, p. 396.)

The late Mr. Barber, of Stamford, communicated to me, in June, 1848, the particulars of a case which shows that even the *flowers* of this plant are highly noxious. A child, between three and four years of age, ate twelve laburnum flowers, and in about fifteen minutes it complained of sickness and severe pain in the stomach. The child vomited a quantity of mucus mixed with the yellow petals of the laburnum. An emetic was given; this cleared the stomach, and the child recovered. There was no purging. (*Guy's Hosp. Reports*, Oct. 1850, p. 219.) A case in which a child suffered from symptoms of a nervous kind by reason of its having eaten laburnum flowers, is described by Mr. North, in the *Medical and Physical Journal*, vol. 62, p. 86.

Analysis.—The bark, flowers, and seeds could be identified only by their botanical characters. A decoction of the bark forms a clear light-brown liquid having an acid reaction. It strikes a dark olive-green color with a persalt of iron. Nitric acid renders it lighter. Acetate of lead precipitates it, but the precipitate has none of the properties of meconate of lead.

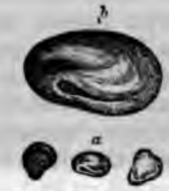
The poisonous principle of the laburnum is called *cytisin*. It is difficult of separation, and at present has no well-defined chemical properties whereby it may be identified. Hence, when administered in powder, infusion, or decoction, there are no chemical processes known by which the poison may be detected. A decoction of the bark forms a clear light-brown liquid, having an acid reaction. It strikes a dark olive-green color with a persalt of iron. Nitric acid renders it lighter. Acetate of lead precipitates it, but the precipitate has none of the properties of meconate of lead. The *leaves* of the laburnum are well known. An illustration of a leaf of its natural size, which is copied from a photograph, is annexed (Fig. 99). The *seeds* are somewhat kidney-shaped, slightly hooked at the hilum. They shrink in drying, become dark-colored, and present irregular depressions on the surface. They have no markings, and are thus easily distinguished from most other poisonous seeds. They are larger than those of datura stramonium (Fig. 100).

FIG. 99.



Leaf of laburnum, natural size.

FIG. 100.



Seeds of laburnum.
a. Natural size.
b. Slightly magnified by a lens.

Cytisin is said to be the poison contained in an insect-powder, which is known by the name of Australian or Persian insect-powder.

YEW (TAXUS BACCATA).

The yew appears to be a cerebrospinal poison. The symptoms produced by the *leaves* and *berries* are uniform in character: convulsions, insensibility, coma, dilated pupils, paleness of the countenance, small pulse, and cold extremities, are the most prominent. Vomiting and purging are also observed among the symptoms. In two cases, the subject of one, a girl about five years of age, died in a comatose state in four hours after she had eaten the *berries;* and the other, a boy, æt. four years, died nineteen days after taking the berries, obviously from severe inflammation of the bowels. The immediate symptoms in the boy were vomiting, purging, coma, convulsions, dilated pupils, hurried respiration, a small pulse, and a cold skin. (See *Prov. Jour.*, November 29, 1848, p. 662, and December 27, p. 708.)

The *leaves* and *berries* of this tree have been long known to be poisonous to cattle, causing death in a few hours, sometimes without vomiting or purging. There is a vulgar but erroneous notion that the leaves are not poisonous when fresh, and that they act only mechanically. It is now well ascertained that yew-leaves and berries exert a specific poisonous action both on men and cattle. If animals recover from the primary effects on the nervous system, they are liable to die after several days from inflammation of the bowels. On one occasion I examined the viscera of an ox which had died from the poisonous effects of yew-leaves. There was much inflammation, and in some parts of the intestines gangrene had taken place.

Symptoms and Appearances—The Leaves.—Dr. Percival states that a tablespoonful of the fresh *leaves* was administered to three children of five, four, and three years of age as a vermifuge. Yawning and listlessness soon succeeded; the eldest vomited a little, and complained of pain in the abdomen, but the two younger children suffered no pain. They all died within a few hours of each other.

In March, 1845, a case was reported to the Dublin Pathological Society by Dr. Mollan, in which a lunatic had died from the poisonous effects of *yew leaves.* The deceased was observed chewing the plant, probably from that perversion of appetite so commonly observed in insanity, and before the attendants had taken it from him he had succeeded in swallowing a portion of the masticated juice. He was soon afterwards suddenly sized with giddiness, prostration of strength, vomiting, coldness of the skin, spasms, and irregular action of the heart. He died in fourteen hours. On inspection, the stomach was found much distended; it contained some yew leaves. There was emphysema in the submucous tissue, but no other abnormal change; there was some thickening with opacity of the arachnoid membrane, which might have been of old standing and due to the insanity. (*Dub. Hosp. Gaz.*, May 15, 1845, p. 109.) A girl, æt. 19, took a strong decoction of the *leaves* to bring on the menses. The dose taken was a tumblerful for four successive mornings. Severe vomiting followed, and this was promoted by tepid water. Delirium came on, and the patient died eight hours after taking the last dose. It is stated that nothing of importance was revealed by an inspection of the body. (*Lancet*, 1870, vol. 2, p. 471.) In another case, reported by Mr. Wallis, in the *British Medical Journal*, a girl, æt. 13, took the leaves for a similar purpose. Death took place rapidly, without any other symptom of poisoning than vomiting. On inspection there was congestion of the membranes of the brain, liver, and kidneys; a greenish color of the contents of the stomach and intestines owing to the fragments of yew leaves, and stellated inflammation of the mucous membrane of the stomach and bowels.

On these occasions it is difficult to obtain any knowledge of the quantity taken. The following case, communicated to me by Dr. Proctor, of York, in May, 1870, shows that the life of an adult may be destroyed by a very small quantity of the fresh leaves. A lunatic woman had been employed in preparing evergreen decorations for Christmas Day. Nothing unusual was observed by the nurses in attendance until about 10 P.M. She had had some bread and cheese with the other patients, when in about five minutes she slipped off her chair almost helpless. Her countenance turned of a dusky pallid hue, but there were no cerebral symptoms. She vomited a quantity of food mixed with a few bits of yew leaves. She soon passed into a state of collapse, and died at 1 A.M.—in less than three hours from her first seizure. She retained her consciousness until a few minutes before she died, and admitted that she had eaten some little bits of yew, but she did not think anything of it. The broken leaflets in the vomited matters and the portions found in the stomach and bowels after death, did not amount to a teaspoonful. Yew leaves may thus prove in small quantity a rapidly fatal poison.

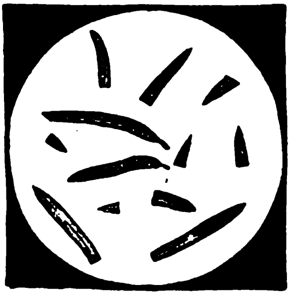
In *Wilson* v. *Newberry* (Queen's Bench, November, 1871), an action was brought against defendant for the loss of two horses by reason of their having eaten yew leaves. The evidence showed that defendant had on his land yew trees, which had been clipped, the clippings having been thrown over the hedge where the plaintiff's horses could have access to them. There was no doubt that the animals had died from eating the yew leaves, but the defendant denied his liability, and the jury returned a verdict in his favor. It was proved that he gave no order for cutting the trees, and he was not aware that they had been cut. On a motion for a new trial (Queen's Bench, June, 1872), judgment was finally given for defendant. Similar evidence of the death of two cows was given in *Lawrence* v. *Jenkins* (Queen's Bench, January, 1873). They strayed through a gap in a hedge into the grounds of defendant, and ate the foliage of the yew trees growing there, from the effects of which they soon died. The question here was, who was responsible; there was no doubt about the cause of death. The question of the poisonous properties of yew on cattle again presented itself in the Rolls' Court in *Erskine* v. *Adams* (March, 1873). In addition to cows, the plaintiff lost a large number of sheep and lambs, by reason of their browsing on yew trees, at the side of a plantation on defendant's farm. Veterinary evidence to this effect was given. These cases should dispel the vulgar error that fresh yew leaves are not poisonous to cattle.

The following case of poisoning by the *berries* of the yew occurred to Mr. Hurt, of Mansfield. A child, aged three years and a half, ate a quantity of yew berries about eleven o'clock. In an hour afterwards the child appeared ill, but did not complain of any pain. It vomited part of its dinner, mixed with some of the berries. A medical man was sent for, but the child died in convulsions before he arrived. On inspection, the stomach was found filled with mucus, and the half-digested pulp of the berries and seeds. There were patches of redness in the mucous membrane, and this was so much softened that it could be detached with the slightest friction. The small intestines were also inflamed.

A lunatic ate a quantity of the *berries* at 10 A.M., and seven hours afterwards he was found dead sitting in a chair. On inspection of the body, the right cavities of the heart were distended with fluid blood of a dirty plum color. The mucous membrane of the stomach was reddened and softened with patches of black congestion. The duodenum was in a similar state. In the lower part of the small intestines there was a mass of the berries. The liver and other soft organs were much congested. (*Med. Times and Gaz.*, 1870, vol. 2, p. 446.) Another fatal case is recorded in this journal for 1871, vol. 1, p. 386.

The nature of the poisonous principle in the yew is unknown, and it is not certain whether, with respect to the berry, the poison is lodged in the pulp or the seed, although it is most probably in the latter. Infusion of yew leaves, which is popularly called yew-tree tea, is sometimes used for the purpose of procuring abortion by ignorant midwives. A case of death from a person drinking this infusion is reported in the registration returns for 1838–9. In the returns for 1840 there is also one death of a woman, æt. 34, referred to her having eaten the berries of the yew. The subject of poisoning by yew leaves, in reference to their employment for purposes of abortion, has been investigated by MM. Chevallier, Duchesne, and Reynal. (See *Ann. d'Hyg.*, 1855, vol. 2, pp. 94, 335.)

Fig. 101.



Yew leaves and fragments, natural size.

Analysis.—Fragments of the leaves or the berries may be found in the stomach. The yew and the savin are the only coniferous poisons which grow in this country. The apex of the leaf of the yew is not so pointed as that of the savin (see Fig. 39, *ante*, p. 480), and the yew leaf does not possess the peculiar odor of savin when rubbed. In the annexed illustration (Fig. 101) the leaf is of the natural size, the engraving having been made from a photograph of the living leaf. Yew berries are seen in autumn; they are about the size of a pea, of a light-red color, dull on the surface, and translucent. They are open at the top, allowing a hard brown kernel to be seen. This is of an ovoid shape, and it forms the greater part of the berry. The fine red skin contains a colorless and remarkably viscid or adhesive juice, which reddens litmus-paper, and has a nauseous sweetish taste.

PRIVET (LIGUSTRUM VULGARE).

The privet is not commonly enumerated among vegetable poisons. No reference is made to this plant in the works of Wibmer, Orfila, Christison, and other writers on toxicology; and yet it would appear, from the subjoined cases—for the brief particulars of which I am indebted to Mr. Ward, of Ollerton—that the *berries* may exert a poisonous action. In December, 1853, three children ate the *berries* of the privet, two of them, a boy three years of age and a girl of six, eating them rather freely. They suffered from violent purging, and when seen by a medical man the little boy was found pulseless and cold, and before death he was frequently and violently convulsed. The girl was in a state of collapse, but rallied a little under treatment; soon afterwards she died convulsed. The surviving child, who had only tasted the berries, did not suffer, and she was enabled to point out the shrub, the berries of which they had gathered. A case has been communicated to me which occurred in November, 1866, in which a child, æt. 2, died thirty-seven days after eating these berries; symptoms of irritation continuing more or less throughout. After death, there were the well-marked appearances of mesenteric disease. According to Loudon, the berries are eaten by birds when other sources of food fail.

Dr. Moore, of Lancaster, has given me a notice of two cases, which show that the leaves of the privet, besides causing vomiting and purging, act upon the brain and spinal marrow. In May, 1872, two children, aged twelve and eight years respectively, ate a quantity of leaves and shoots, proved subsequently to have been those of the privet. The symptoms in both cases were drowsiness, convulsive twitchings, difficulty in moving about, loss of muscular power, severe vomiting and purging; the evacuations being of a greenish color. They both recovered.

THE HOLLY (ILEX AQUIFOLIUM).

From some facts recently published, the red *berries* of this tree appear to produce the effects of narcotico-irritant poisoning. A boy, three years old, ate a number of them. The symptoms which followed were sickness, pain in the head and abdomen, with much purging. Many of the berries of the common holly were passed in the motions; drowsiness supervened, and there was loss of consciousness. In this state (after twenty-four hours) he was seen by Mr. Barkas. His face was pale; the skin pale and cool; pulse weak and small (80). The pupils of the eyes were much contracted, but were sensible to light. The vomiting had ceased, but there was some purging. Castor oil and stimulants were given, and on the second day the child recovered. (*Lancet*, 1870, vol. 1, p. 573.) Wibmer speaks of these berries as having merely a purgative action.

GUELDER ROSE (VIBURNUM OPULUS).

The noxious properties of this plant have received but little notice. Wibmer speaks of its flowers and berries as having acrid properties, and Lindley describes the plant, generally, as emetic and purgative. This vegetable is not, however, simply irritant to human beings; it has manifested an action on the brain and nervous system.

In October, 1870, five children in a family at Sudbury suffered from symptoms of poisoning as the result of eating the white *berries* of this shrub, commonly called snow-berries. Mr. W. B. Smith communicated to me the particulars. One Sunday morning the five children were simultaneously seized with violent vomiting, which lasted for many hours. Mr. Smith saw them on Monday morning. The vomited matters had then been thrown away. One girl, æt. 5, was in a state of profound coma and insensibility; pupils not much dilated; pulse 40; legs rather rigid; the arms not at all so. This girl died at 8 P.M., about thirty-six hours after eating the berries. Another child suffered from similar symptoms, but in a less degree, and recovered in two or three days.

In the case of the child that died, the stomach and intestines were quite empty; there were no marks of inflammation. The brain was slightly congested on its surface, but not in its substance, and there was no effusion. This is the only instance of poisoning by this plant that I have met with.

QUINOIDINE.

This is a dark resinoid uncrystalline substance contained in the mother-liquors from which the salts of quinia have been extracted. It has been used medicinally as a substitute for these salts. Its properties are analogous to those of quinia. As a poison it is but little known. Dr. Tidy has reported the following case. A man employed in some chemical works, thinking that he was taking an aperient mixture, swallowed two ounces of an "Ague mixture," containing about eighty-five grains of quinoidine in each ounce, making a dose of one hundred and seventy grains of quinoidine. He vomited violently immediately after taking the medicine, and died in about half an hour. On inspection, the principal appearances were congestion of the brain, with a generally congested state of the stomach. This organ contained a brown liquid in which quinoidine is said to have been detected. (*Lancet*, 1872, vol. 2, p. 41.) From experiments on animals this substance appears to act on the brain and alimentary canal. It is a cerebral and irritant poison. (Husemann's *Pflanzenstoffe*, 1871, p. 355.) It has caused in dogs, salivation, vomiting, great depression, drowsiness, tremors of the head and body, and in fatal cases tonic and clonic convulsions. It destroyed life in from four to six hours. With the exception of congestion of the brain, nothing was found on inspection.

CURARA AND CURARINA.

According to Schomburgk, the tree which produces curara grows in Guiana. It furnishes a poisonous juice or extract, which, when mixed with other substances, forms an arrow-poison used by the Indians in killing game, or destroying each other. Various names have been given to the extract, according to the district in which it is prepared; but from the recent investigations of Bernard and Pelikan, it is evident that the poison known under the name of *Woorali*, *Oorara*, and *Curara* does not owe its effects to strychnia, and that the plant or plants which yield it do not belong to the strychnos tribe. At the same time, among the variety of poisonous extracts used by the Indians, there may be one or more containing strychnia. Martius affirms that the Ticunas extract is derived from the *Cocculus Amazonum*, and that it contains picrotoxin.

The South American poison is now generally known under the name of Curara, from the plant *Curari*, from which it is obtained. It contains a poisonous alkaloid first discovered by Boussingault, in 1828, which is called *Curarina*. Animals have been said to fall instantly dead when shot with an arrow poisoned by Curara, but this has only been in cases where a vital organ like the heart has been directly wounded, and then death was not due to the poison. According to Mr. Waterton, the poisonous extract is procured chiefly from the bark of a creeper or vine which grows in the forests of Guiana and Central America. The Indians prepare the poison with a great deal of mystery, and mix with it other herbs, red and black ants, and the pounded fangs of a venomous snake. The juice is extracted from the stem of the creeper by infusion and compression; it is then heated with the other ingredients over a slow fire until it acquires a dark-brown color, and an intensely bitter taste. It is afterwards put into a small pot, carefully covered over and kept in a dry place. It is occasionally warmed over a fire that it may be kept dry.

The extract is miscible with water, and when fresh the slightest moisture dissolves it; hence, it speedily diffuses itself when introduced into a wound. The symptoms which it produces in animals are stupor and paralysis. It does not begin to produce any apparent effects until after a lapse of one or two minutes, and there is apparently no pain; convulsions come on in two or three minutes, and the animal dies in four or five minutes. Putrefaction is not accelerated, and the flesh of the game thus killed is used as food without any serious effects resulting. This is probably due to the very small quantity of absorbed poison present; for the Curara, in a sufficient dose, is fatal to all animals. It requires much more of this extract to kill an ox than a smaller animal; thus, the Indian adjusts the size of his arrow and the quantity of poison to the size of the animal. In one experiment, three arrows were introduced beneath the skin of an ox. For four minutes there was no effect: the animal then set itself firmly on its four legs as if to resist falling, and remained quite still for fourteen minutes. It then attempted to walk, staggered, and fell. The eyes became fixed, dim, and apparently insensible to light. Convulsions appeared in the legs; there was emprosthotonos, laborious respiration, and an escape of a frothy liquid from the mouth. The convulsions in the extremities gradually ceased; there was still a perceptible action of the heart at intervals. In twenty-five minutes the animal was quite dead. The flesh was eaten, and gave rise to no unpleasant symptoms, nor was it observed to have any peculiar taste. The poison does not appear to have any action on the heart, for that organ continues to pulsate after respiration has ceased. From one grain and a half to two grains sufficed to kill rabbits, and a smaller quantity operated fatally by hypodermic injection.

There is no known antidote to the effects of Curara when it is once absorbed into the blood. As in reference to serpent-poison, the application of a ligature between the wound and the heart and an early and free excision of the part, present the only chance of safety. Mr. Iliff states that he found the extract to retain its poisonous properties for a period of twenty-seven years (*Med. Gaz.*, vol. 20, p. 282); but unless kept dry it is liable to become weakened. Bernard found that some which had been loosely kept on the tip of an arrow for fifteen years killed an animal very quickly. He preserved it in a state of solution in water for two years without any loss of its power. (*Leçons*, p. 258.)

Curara, according to the experiments of Bernard, is, like the serpent-poison, active when introduced into a wound, but almost inert when taken into the stomach. (*Leçons sur les Effets des Substances Toxiques*, Paris, p. 239.) Small animals are killed in a few minutes when the poison is injected into a wound: they lose all power over the muscles, become insensible, and die without convulsions. Kölliker found that it scarcely affected the spinal marrow, but that it paralyzed the voluntary muscles. Pelikan observed that the alkaloid *curarina* operated in a similar manner. Its effect is to destroy the motor power of the nervous system. Dr. Fraser found that curara completely paralyzed the motor nerves. (*On the Calabar Bean*, p. 271.) These results show that it operates in the reverse manner to strychnia, and that curarina and strychnia are completely antagonistic. An account of the properties of this poison will be found in the *Ann d'Hyg.*, 1866, vol. 2, p. 155, by MM. Voisin and Lionville, and MM. Tardieu and Roussin, *L'Empoisonnement*, p. 380.

Analysis.—Curara is a brownish-black-looking brittle substance, having the appearance of Spanish liquorice. It dissolved slowly in cold water, but rapidly when heated, producing a turbid brown liquid. This became clear on filtration, and possessed the following properties. It was quite neutral—readily precipitated by the chloriodide of potassium and mercury, the ioduretted iodide and tannic acid—showing its alkaloidal character. Iodic acid produced no change in it. Nitric acid gave to the solution a dark red-brown color. Sulphomolybdic acid produced a slate-gray tint. It may be regarded as a dry organic substance, containing the alkaloid curarina.

Curarina.—The properties of curara are due to the presence of this alkaloid. It is soluble in water, alcohol, and acids and alkaline liquids. The aqueous and alcoholic solutions have a rich red color, and an intensely bitter taste. Curarina has hitherto been procured only in small quantities as a dry uncrystalline solid. It is alkaline in its reaction, neutralizes acids, and produces salts which do not crystallize. When heated, it evolves thick vapors, which have an intensely bitter taste. Strong nitric acid produces with it a blood-red color; sulphuric acid gives with it a rich carmine tint, in which characters it resembles brucia. On the other hand, MM. Pelikan, Voisin, Tardieu, and Roussin have found that pure curarina possesses the chemical properties of strychnia so far as the color-tests are concerned, the sole difference being that curarina produces a blue color with sulphuric acid alone. The galvanic test acted similarly on both alkaloids. (Bernard, *op. cit.*, p. 474.) M. Bernard remarks that this similarity of chemical results proves that there is no direct relation between the chemical characters of a substance and its physiological effects. Similar chemical characters may exist in two bodies (curarina and strychnia) of which the physiological effects are not only different, but antagonistic. M. Voisin found some difficulty in separating curarina from the viscera of animals poisoned with it, but the urine of such animals was found to operate by hypodermic injection on other animals with the usual symptoms of curara poisoning. (*Op. cit.*, p. 158.)

CEREBROCARDIAC POISONS.

CHAPTER LXXVI.

Action of poisons on the heart and brain—The Borneo and Java poisons—Upas antiar—Antiarin—Tanghinia—The kombi—Cobra poison—Calabar bean—Physostigmia.

Among the neurotic poisons there are some which especially act on the *heart*. They reduce its pulsations, paralyze it, and destroy life by syncope. They may also exert an action on the brain and spinal marrow. Some of the poisons of savage tribes possess this peculiarity. Dr. Braidwood has described one of these from Borneo, the *Dajaksch*, where it is used as an arrow-poison. It is in the form of a brittle extract, of a dark iron-gray color, dissolved by water, but not so readily as curara. The solution has a brown color, an alkaline reaction, and a bitter taste. It is insoluble in chloroform. Its action, as determined by absorption through wounds in the skin of animals, consists in paralyzing the heart and stopping its contractions by inducing perfect paralysis of the cardiac ganglia of the sympathetic nerve. Hence it causes an entire destruction of motion and sensation, and the animal suddenly falls dead. Like the calabar bean and opium, it produces contraction of the iris. (*Ed. Monthly Jour.*, Aug. 1864.)

There are other cardiac poisons in use among savage nations. The Upas Antiar, according to Pereira, is derived from a large forest tree in Java (Antiaris toxicaria), growing to the height of from 60 to 100 feet. The milky juice contains 3.56 per cent. of a poisonous principle, called *Antiarin*. It was long since pointed out by Sir B. Brodie that this poison operated by paralyzing the heart. Kölliker and Pelikan have investigated the subject, and have arrived at a similar conclusion; but they found that its principal action was on the voluntary muscles, and that it was essentially a paralyzing poison. It destroys the excitability of the nervous system instead of exalting it like strychnia. It acts with great rapidity on the heart, stopping its action in five or ten minutes. These results have been more recently confirmed by the observations of Dr. Braidwood. He found that it acted directly on the muscular fibres of the heart, and not on the cardiac ganglia like the Borneo poison. It differs from it both physiologically and chemically.

The *Tanghinia Venenifera*, or Madagascar poison, is a seed of a brownish-black color, of the size of an almond, presenting a wrinkled surface, with an odor resembling that of violets. The fruit resembles the almond, but is larger. This is used in Madagascar as an ordeal poison. Its effect, according to Kölliker, is to paralyze the heart.

The *Kombi* arrow-poison of Africa has been described by Dr. Fraser as acting primarily on the heart, producing cardiac paralysis. It also acts on the voluntary muscles, by which their activity is gradually impaired, and finally completely destroyed. (*Proc. R. S. Ed.*, 1869-70, p. 102.) In the last property it resembles curara.

The *venom* of the Cobra de Capello occasionally operates as a cardiac poison. This happens, according to Dr. Fayrer, when the poison is introduced into a wound in large quantity, when it has been rapidly absorbed, or when injected into the jugular vein of an animal. In all these cases, the action of the heart is at once arrested. This does not appear to be owing to paralysis, but to tetanic contraction of the organ from excessive stimulus. In small quantities it paralyzes the voluntary muscles.

There are several other organic poisons which manifest a strong action on the heart, and in some cases produce death by paralyzing this organ. Among these may be mentioned chloroform, hydrate of chloral—both of which depress the action of the heart and cause death by syncope—and aconite. The ordinary mode of action of these substances, however, justifies their position among the cerebral and cerebrospinal poisons. Again, some mineral poisons, although properly classed and described as irritants, manifest a direct action upon the heart. Tartar emetic is well known to exert a depressing action on the circulation, and in this form of poisoning, death may sometimes take place suddenly from syncope.

The first of the poisons in this group which claims notice is the Calabar bean, an ordeal poison of Old Calabar, on the West Coast of Africa.

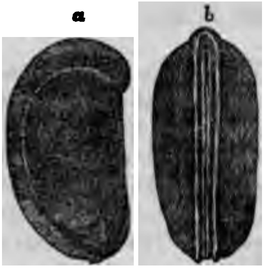
CALABAR BEAN (PHYSOSTIGMA VENENOSUM).

The Calabar bean is a large leguminous seed of a dark color, resembling a large horse-bean, but much thicker and more rounded in its form. It is the seed of the *Physostigma venenosum.* It is brought from the western coast of Africa, and is there employed by the natives as an ordeal bean when persons are suspected of witchcraft. The common belief is that innocent persons who take it, vomit, and are safe, while the guilty retain the poison and die from its effects. So strong is popular confidence in this test, that those who are suspected, voluntarily take an emulsion of this dreadful seed; and, as Sir R. Christison remarks, many an innocent person thus pays the penalty of his rash reliance on a superstitious custom. As it is a firm matter of faith that if a man dies he is guilty, such a custom is beyond the reach of any appeal to reason. Illustrations of this bean of its natural size are annexed. (See Fig. 102, p. 756.)

This bean owes its properties to the presence of an alkaloidal substance called *Physostigmia.* It is found in the cotyledon, and the complex process adopted for its separation by Jobst and Hesse is described in the *Chemical News* for March 5, 1864, p. 109. The medicinal dose of the powdered bean is from one to four grains. The dose of the extract, which is made with rectified spirit, is from one-sixteenth to one-quarter of a grain.

The bean or seed has a thin, hard, dark-colored, brittle covering; the kernel inside is white, and weighs from thirty-six to fifty grains—the whole seed about sixty-seven grains. (Fig. 102.) Sir R. Christison could detect no poisonous alkaloid in the seed, but he found that the active principle (physostigmia) could be extracted by alcohol, which dissolves 2.7 per cent. of the seed, including this substance. That alcohol will remove the poisonous principle is proved by the fact that the exhausted residue is not always poisonous. (Bouchardat, *Ann. de Thérapeutique*, 1864, p. 73. See also *Pharm. Jour.*, 1863, p. 561.) The greater part of the seed, as in nux vomica, consists of inert substances, with a small quantity of oil. The kernel is yellowish-white, without bitterness, acrimony, aroma, or any other impression on the organ of taste. In fact it cannot be distinguished by taste from a haricot-bean.

FIG. 102.



a. The bean, of its natural size.
b. The same, seen edgewise.

Symptoms and Effects.—Twenty-one grains in fine powder were placed in the cellular tissue of a rabbit; for three minutes there was no change. The animal then became weak and languid; in four minutes it was unable to raise itself when placed on its side. The body then became quite flaccid, and respiration ceased in five minutes. There were at intervals slight irregular twitchings in the muscles of the trunk, and a jerking of the head backwards. *Two grains* of the alcoholic extract produced similar symptoms. At the end of two minutes, without any previous indication, the animal suddenly became weak, fell on its side, struggled a little with its feet, and ceased to breathe in another minute. The poison, according to Sir R. Christison, produces a primary impression on the heart, causing paralysis of that organ, the insensibility and coma being only apparent. The results appear to show that there is also paralysis of the voluntary and respiratory muscles, with a retention of consciousness. (*Pharmaceutical Journal*, 1855, p. 473.)

Desiring to try the effects of this seed on himself, Sir R. Christison took the eighth part of a seed, or six grains, one night before going to bed. There was a slight sense of numbness in the limbs during the night, but in the morning no urgent symptoms of any kind. He then chewed and swallowed the fourth part of a seed (twelve grains): In twenty minutes he was seized with giddiness, and a general feeling of torpor over the whole frame. He immediately swallowed an emetic, and thus emptied his stomach. The giddiness, weakness, and faintness increased to such a degree that he was obliged to lie down in bed. In this state he was seen by two medical friends, who found him prostrate and pale, the heart and pulse extremely feeble and tumultuously irregular, the mental faculties entire, extreme faintness threatening dissolution, but no apprehension of death on the part of the patient. There was no uneasy feeling of any kind, no pains or numbness, no prickling, not even any sense of suffering from the great feebleness of the heart's action. There was the will but not the power to vomit; the limbs became chill with a vague feeling of discomfort. Stimulants were employed, and warmth and pulsation, with a power of moving, gradually returned. Two hours after the poison had been taken he felt drowsy, and slept for two hours more, but with such activity of mind that he had no consciousness of having been asleep. The tumultuous action of the heart continued. After this the symptoms gradually disappeared, and the next day he was quite well. (*Pharm. Jour.*, 1855, p. 474.) Physostigmia does not act so much upon the brain as on the spinal marrow. While laboring under the effects of the poison, Sir R. Christison maintained his mental vigor, but the pulsations of the heart were greatly reduced.

In April, 1864, two children, aged 6 and 3 years respectively, chewed and ate the broken fragments of the kernel of one nut. In about forty minutes they complained of sickness. One child held his head drooping, appeared sleepy and his hands were powerless. He staggered and was scarcely able to walk. He complained of severe pain in the stomach, and made ineffectual attempts to vomit. Milk was given, and he then vomited. The child became quite prostrated, the pulse was feeble and slow, and the pupils were slightly contracted. Some pieces of the nut were thrown up by vomiting. The other child had pain in the abdomen, and was listless, sleepy, and depressed. He vomited freely, some portions of nut being ejected. He could neither stand nor walk. His face was pale, the eyes were piercing, but the pupils and pulse were natural. In this case there was purging. The children recovered on the third day. (*Edinburgh Monthly Journal*, 1864, p. 193.) In August,1864, from fifty to sixty children were poisoned at Liverpool by reason of their having eaten these beans. The sweepings of a ship from the West Coast of Africa had been thrown on a heap of rubbish; the children found the beans and ate them. In two hours forty were brought to the hospital. Thirty suffered from violent retching. One of them, æt. 6, who had eaten six beans, died soon after his admission. The principal symptoms from which he suffered were severe griping pains, incessant vomiting, and contracted pupils. The stomach-pump and emetics were employed with great benefit. When admitted, the children were pale, very sick, and exhausted, and when they attempted to walk they staggered about as if they were drunk, although they had the use of all their senses, the poison evidently not producing that stupefying effect which results from the taking of opium. Their pulses were at first very low; some of them became feverish and drowsy, and their eyes were bright and protruding from the sockets, and in some of the worst cases, the pupil of the eye was contracted. The quantity of beans taken could not be correctly ascertained. In cases in which it has proved fatal to animals it has caused much irritation and congestion of the stomach and bowels. (Dragendorff.)

A drop of the extract applied to the eye, produces in from ten minutes to a quarter of an hour a remarkable contraction of the pupil. This has been observed to last in children for fifteen to twenty hours. (Bouchardat, *Ann. de Thérapeutique*, 1864, p. 73.) In this respect, the poison is eminently distinguished from atropia, daturia, and hyoscyamia, which cause excessive dilatation of the pupil. Dr. Harley found in his experiments with this substance that it caused contraction of the pupil when taken internally, as well as when applied locally. It paralyzed the motor nerves, and left the intellect and muscular irritability unimpaired. It destroyed life by paralyzing the respiratory muscles, and although, according to him, it weakened the heart's power, it neither stopped the circulation nor arrested the heart's action. It is not, therefore in his view, a cardiac, but a respiratory poison. It is closely allied in its effects to curara and conia, but more to the latter. It differs from both in its tendency to produce muscular twitchings, and in its power of causing contraction of the pupil. Neither curara nor conia has any specific effect on the iris. (*Lancet*, 1863, vol. 1, p 717.) The physiological action of this substance has been fully investigated by Dr. T. Fraser. (*Trans. of R. S. Edinburgh*, vol. 24, 1867.)

There are some remarkable circumstances connected with this action on the iris. The pupil of that eye only is affected to which the extract has been directly applied. By a very ingenious experiment, Dr. Fraser has shown that this local and unilateral action does really depend on absorption, and that it probably arises from direct contact of the poison with the iris. He found that the contraction was caused much more rapidly and was maintained for a longer time than when it had been produced as a symptom in cases of poisoning with this substance. In order to prove the presence of the poisonous principle within the eyeball to which it had been applied, he removed the aqueous humor of the eye and placed it on the conjunctiva of another animal. The usual effects of the substance in causing contraction of the pupil were produced. There is then a local as well as general absorption, and so limited is the range of this that in the same animal contraction of the pupil from calabar bean may exist in one eye, while dilatation by the action of belladonna may be produced in the other. (*On the Physiological Action of the Calabar Bean*, 1867, p. 67.) The effect of this substance in contracting the pupil of the eye has been witnessed by Borelli as long as six hours after death; in exceptional cases even so late as twenty-four hours after death; hence, this is not a test of death. (*Am. Jour. Med. Sci.*, April, 1872, p. 582.)

Analysis.—Physostigmia combines with acids to form salts. Dragendorff found that a solution of bromine in water acted in a characteristic manner on a solution of the sulphate, even when diluted to $\frac{1}{10000}$th part. It produced a red color when less than the 1000th of a grain was present. The chloriodide of potassium and mercury also precipitates physostigmia in a much diluted state. The physiological test consists in the application of a solution or alcoholic extract to the eye. It produces strong contraction of the pupil when this liquid contains but a small fractional proportion of physostigmia. Dragendorff has found that it is absorbed and diffused throughout the body. He has separated it by means of benzole used as in separation of strychnia by ether. It is rapidly eliminated by the saliva and other secretions under putrefaction. (Husemann's *Jahresbericht*, 1872, p. 570.)

CHAPTER LXXVII.

FOXGLOVE—SYMPTOMS AND EFFECTS—FATAL DOSE—TREATMENT—DIGITALIA—ITS CHEMICAL AND PHYSIOLOGICAL PROPERTIES—CRIMINAL ADMINISTRATION OF IT—TOBACCO—SYMPTOMS AND APPEARANCES—LOCAL ACTION—NICOTINA—ITS CHEMICAL AND PHYSIOLOGICAL PROPERTIES—FATAL DOSE.

FOXGLOVE (DIGITALIS PURPUREA).

PURPLE foxglove is a well-known hedge plant growing abundantly in the southern districts of England. All parts of the plant, the seeds, leaves, and root, are poisonous owing to the presence of the poisonous principle *digitalin.* The leaves, whether in the form of powder, infusion, extract, or tincture, exert an action on the brain, spinal marrow, and heart, as well as on the stomach and bowels. They retain their noxious properties when dried.

Symptoms and Effects.—Cases of poisoning by foxglove are not very numerous. One was the subject of a criminal trial at the Old Bailey in Oct. 1826. A quack was indicted for the manslaughter of a boy under the following circumstances. He prescribed for a trivial complaint six ounces of a strong decoction of the *leaves.* The boy was soon attacked with vomiting, purging, and severe pain in the abdomen. After some time he became lethargic, and slept for several hours; in the night he was seized with convulsions. The pupils were dilated and insensible, the pulse was slow, small, and irregular; coma followed, and the boy died twenty-two hours after taking the poison. On inspection, the membranes of the brain were found much injected, and the mucous lining of the stomach was partially inflamed. The prisoner was acquitted of the charge, because he had given his advice only on the application of the friends of the deceased! (*Ed. Med. and Surg. Jour.*, vol. 27, p. 223.) For cases of recovery from a strong dose of the infusion, see *Med. Gaz.*, vol. 34, p. 659; and *L'Union Médicale*, 24 Août, 1848. On the other hand, a case in which an infusion of the *root* proved fatal, is reported in the *Lancet*, July 14, 1849, p. 31. Accidents sometimes occur from the medicinal use of the *tincture.* In one case, in which a dose of the tincture was too frequently repeated, the person was attacked with restlessness, thirst, inflamed eyes, and other serious symptoms.

A young man having filled a quart pitcher with the leaves of foxglove, poured upon them as much boiling water as the pitcher would hold. Of this strong *infusion* he took a teacupful on going to bed, which caused him to sleep soundly. In the morning he took a second cupful (the infusion being much stronger), and he then went to his employment. He soon felt dizzy and heavy, began to stagger, lost his consciousness, and at length fell down in a state of syncope. On being conveyed home he vomited severely, and suffered great pain in the abdomen. When visited he was conscious, and complained of pain in his head; the pupils were dilated, the skin was cold, pallid, and covered with a copious perspiration. The pulse was low, about forty in the minute—three or four feeble pulsations being succeeded by a complete intermission of several seconds; and each stroke, though weak, was given with a peculiar "explosive shock." There was still great pain in the abdomen, with incessant and violent vomiting, no purging; suppression of urine, and an abundant flow of saliva. Brandy and ammonia with warmth were employed, and after reaction had commenced purgatives were administered. The man slowly recovered, but the pulse presented its peculiar beat and weakness for several days; and during this time the man could not bear the upright position.

In another instance, a young man swallowed a strong *decoction* of foxglove by mistake for purgative medicine. He was soon seized with vomiting, pain in the abdomen, and purging. In the afternoon he fell asleep. At midnight he awoke, was attacked with violent vomiting, colic, convulsions, dilated and insensible pupils; and his pulse was slow and irregular. He died twenty-two hours after taking the poison. (Wibmer, *op. cit.*, *Digitalis.*) A few grains of the powdered leaves have been known to produce giddiness, languor, dimness of sight, and other nervous symptoms. A drachm has, however, been taken without causing death; but in this instance it produced the most violent vomiting. As indicative of the singular effect of the poison on the nerves of sensation, it may be stated that a coal fire appeared to the patient to have a blue color. A common effect of this poison is to produce great depression of the heart's action, showing that it is a cardiac poison.

A woman made an infusion of digitalis, and swallowed it by mistake. The symptoms which followed were vomiting, paleness of the face, coldness of the skin, prostration, muscular feebleness, a persistent feeling of drunkenness, headache, giddiness, confusion of sight, dilatation of the pupils, and loss of sensibility. The vomiting was constant, and aggravated by anything that was taken. There was constipation of the bowels, with suppression of urine. There was thirst, with pains in the abdomen, increased by pressure, and great restlessness at night. At first the pulse was 52. On the fourth day it was 41 to 42. On the fifth day it was 58, less irregular, and the symptoms had abated. During the night she got up, and on returning to her bed suddenly fainted, and died. Nothing could restore her from the attack. This was probably the effect of the poison on the heart. In order to avoid death by syncope a person laboring under symptoms of poisoning by digitalis should always be kept in the recumbent posture. (Case by Dr. Mazel, *Ed. Monthly Jour.*, 1864, p. 169.)

In the case of a man, æt. 50, the *tincture*, taken in medicinal doses for about twenty days, produced the following train of symptoms. (*Med. Gaz.*, vol. 31, p. 270.) The pulse, which, during a former use of the medicine, had been reduced by ten or fifteen beats in a minute, sank almost to half its usual number. The patient was tormented by the most painful disquietude, so that, even in the night, he left the bed every moment, could not sleep, and with his eyes open conversed with persons who were not present. At the same time the pupils were dilated, the conjunctiva both of the eye and the lids was red; he had but little appetite, with great nausea, violent thirst, and dryness of the mouth; the alvine evacuations were scanty; the secretion of urine was increased. These phenomena, which obviously were merely the effects of the digitalis, had lasted six days, when the restlessness diminished, sleep returned, and the dilatation of the pupils disappeared. This case shows that digitalis possesses accumulative properties; and that it cannot be given for a long period medicinally without producing dangerous symptoms. The late Dr. Elliotson observed that persons who had been in the habit of taking foxglove in medicinal doses for a long period died very suddenly from syncope, as if a fatal impression had been produced on the heart by the accumulation of the poison in the system. The *appearances* which have been met with after death, are congestion of the brain and its membranes; inflammation of the mucous membrane of the stomach, and fluidity of the blood.

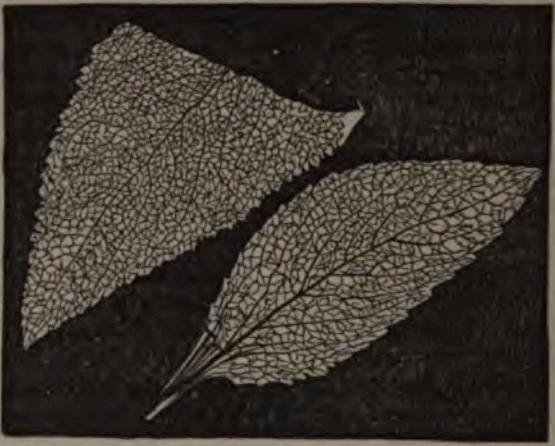
Fatal Dose.—The medicinal dose of the infusion is from two to four drachms; of the tincture from ten to thirty minims; and of the powder from half a grain to one grain and a half. The medicinal preparations vary considerably in strength, a fact which will explain why they have been administered in much larger doses than those here assigned, without producing dangerous effects. According to the late Dr. Pereira, twenty drops of the tincture were given to an infant, laboring under water on the brain, three times daily for a fortnight, without causing any untoward symptom; and he frequently prescribed for an adult one drachm of the tincture three times daily for a fortnight without producing any marked effect. The tincture has been sometimes prescribed medicinally in doses of half an ounce to an ounce; and on one occasion two ounces were taken in two doses without giving rise to the slightest inconvenience. These facts show either that foxglove, as a vegetable, is not so powerful a poison as it is commonly supposed to be, or that the proportion of digitalin is liable to great variation in the alcoholic solution.

Treatment.—In a case of poisoning by foxglove, in addition to the free use of emetics, vegetable infusions containing tannic acid should be given. According to the researches of M. Homolle, this renders the active principle insoluble.

Analysis.—When foxglove has been taken in substance, *i. e.*, in the form of seeds or leaves, or any portion of these has been swallowed in a decoction or infusion, fragments may be found in the stomach and bowels. In reference to the infusion, decoction, tincture, or extract, except there be sufficient to allow of the separation of digitalin, there is no chemical process known by which the poison may be recognized. If any fragments of leaves or seeds are found in the contents of the stomach or in food, they may be identified by the aid of the microscope. The annexed illustration (Fig. 103, p. 762), taken from the living plant by photography, represents a small leaf and a portion of a larger leaf of the purple foxglove.

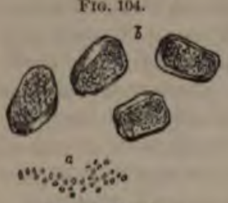
It is calculated that digitalin constitutes only one per cent. of the dried leaves. In reference to the *seeds* of foxglove, they are of a reddish-brown color, remarkably small, oblong, and somewhat angular in shape. They have peculiar markings (Fig. 104). By the aid of the microscope they may be easily distinguished from the seeds of hyoscyamus, datura, belladonna, and most other poisonous plants.

FIG. 103.



Leaves of foxglove.

FIG. 104.



Seeds of foxglove.
a. Natural size.
b. Magnified 30 diameters.

DIGITALIN.

The active principle of foxglove is called *digitalin* or *digitalia*. Its properties have been investigated by M. Homolle. (*Journ. de Pharmacie*, Janvier, 1845, p. 57; also Bouchardat, *Ann. de Thérap.*, 1864, p. 155.) The process for obtaining it is exceedingly complex. It is an inodorous, imperfectly crystalline substance of a pale fawn color. It is so intensely bitter that it gives a sensible bitterness to 200,000 parts of water; but the taste of digitalin itself is only slowly manifested, in consequence of its great insolubility. It requires 2000 parts of cold, and 1000 parts of boiling water for its solution. It is much more soluble in alcohol and ether, but alcohol is its best solvent. When dissolved in either menstruum, it has neither an acid nor alkaline reaction. It is a neutral vegetable principle, forming no salts with acids, hence *digitalin* is a more appropriate name.

M. Nativelle has recently obtained it by the aid of boiling alcohol at 90 per cent. in fine white and shining needles. (*Pharm. Jour.*, 1872, April 27, p. 865.) In solution, digitalin is precipitated by tannic acid; but not by the chloriodide of potassium and mercury; and by this property it may be distinguished from the true vegetable alkaloids. When heated in a tube, it melts and is decomposed; it evolves an acid, and not an ammoniacal vapor. Strong nitric acid dissolves it, and gives to it a yellowish-brown color, in which it differs from morphia and brucia. This acid solution speedily acquires a pale yellow color on standing. Iodic acid is unchanged by it. Hydrochloric acid dissolves it, and when gently heated the solution becomes green. Strong sulphuric acid gives to it a reddish-brown color, and after exposure for some time or by a gentle heat this color changes to a purple-black. If the sulphuric acid solution is diluted, the liquid immediately assumes a dingy green color. Diluted sulphuric acid heated with the powder gives a reddish-black color. Sulphomolybdic acid produces with it a dark purple color. M. Grandeau has suggested an addition to this test. If the digitalin has been previously dissolved and the liquid evaporated, sulphuric acid imparts a rose-color to small quantities, or a reddish-brown or even brown color when the digitalin is in rather large quantity. If the digitalin, moistened with sulphuric acid, is exposed to the vapor of bromine, it immediately assumes a violet color. This peculiar color is observed even with the faintest trace of the principle, and it is regarded by him as characteristic. Seventeen of the alkaloids and principles thus tested did not acquire a violet color. (*Chemical News*, July 16, 1864, p. 26.) According to M. Grandeau, digitalin readily admits of separation by dialysis from organic liquids. MM. Tardieu and Roussin have not found this method so successful in practice as these experiments on pure digitalin would appear to indicate. (*Ann. d'Hygiène*, 1864, p. 80.)

Symptoms and Effects.—M. Homolle extracted from foxglove, by means of alcohol, three substances: an acrid matter soluble in ether, which produced on himself violent vomiting and fearful head-symptoms leading almost to the destruction of life; a highly concentrated bitter principle; and digitalin. The latter alone was proved upon hospital patients to have the power of decreasing the action of the heart, lowering the pulse, and increasing the quantity of urine. (*Pharmaceutical Journal*, Oct. 1861, p. 245.) Pure digitalin itself operates powerfully on man and animals in very small doses, and it must be regarded as a deadly poison. The $\frac{1}{18}$th of a grain, which is considered to be equal to eight grains of the well-prepared powder of the dried leaves, is sufficient to cause symptoms of poisoning. Doses of from $\frac{1}{11}$th to $\frac{1}{32}$d part of a grain have lowered the pulse and caused nausea, vomiting, griping, purging, and an increased secretion of urine. (Pereira, *Mat. Med.*, vol. 2, p. 528.) Doses of from one-quarter to one-half of a grain would probably prove fatal to life. M. Homolle found in experiments on himself that small doses of digitalin taken at intervals lowered the pulse to about one-fourth or one-fifth of the normal standard: thus in himself it fell 17 in one minute; this represents a fourth of the normal pulsations. In doses of from $\frac{1}{15}$th to $\frac{1}{30}$th of a grain in twenty-four hours, digitalin slackened the circulation. In doses above $\frac{1}{15}$th of a grain, it produced on adults emetic and purgative effects, sometimes suddenly, at others slowly and gradually. In doses of from one to two grains, unless speedily thrown off by vomiting, it killed dogs in a few hours. (Orfila, *Toxicologie*, vol. 2, p. 350. See also a paper by Drs. Stevenson and Fagge, *Guy's Hosp. Rep.*, 1866, p. 37.) These gentlemen have fully investigated the action of this poison on frogs. They find that it produces speedily irregularity and stoppage of the heart's action, and that voluntary power is retained for at least fifteen or twenty minutes after the heart has ceased to beat. (*Guy's Hosp. Rep.*, 1866, p. 80.)

Digitalin has acquired some notoriety by reason of the trial of Dr. De la Pommerais, at Paris, in May, 1864, for the murder of a woman named Pauw. The deceased, who was about forty years of age, and in the enjoyment of good health, was suddenly seized with violent vomiting, and after an illness of about twenty-four hours, died on the 17th November, 1863. The prisoner had just renewed his intimacy with her at the time of the occurrence of this fatal illness, and after a long interval of absence; he had induced her to insure her life in various insurance offices for an enormous sum of money, quite disproportionate to her circumstances. Immediately after her death, he put in a claim for these large insurances. The body of the deceased was exhumed and inspected for the first time on the 30th of November, thirteen days after death. The viscera throughout were healthy; they presented no unusual appearance, and revealed no natural cause of sudden death. The stomach and bowels, which were well preserved, bore no marks of the action of poison; and on a chemical analysis, no poison of any kind could be detected in these organs by MM. Tardieu and Roussin. The symptoms, during the illness, owing to there being no suspicion of poisoning, were not accurately observed. Repeated vomiting, with great depression and exhaustion, seem to have been the most prominent. Failing to detect any poison by chemistry and the microscope, the experts adopted the physiological test of administering prepared alcoholic and aqueous extracts of the stomach and intestines to animals. An attempt made to separate the active principle and remove the organic matter by dialysis, did not yield satisfactory results. (*Annales d'Hygiène*, 1864, vol. 2, p. 105.) Seventy-five grains of the mixed extracts above mentioned were introduced into the cellular membrane of the thigh of a dog. The animal vomited twice; and in four hours the pulsations of the heart sank from 102 to 86; its action was irregular and intermittent, and the respiration was deep and painful. There were no narcotic symptoms; on the next day, the dog was better, and it completely recovered. Sixty grains of these extracts in water, administered to a rabbit by means of a funnel, caused death in a few minutes, probably from syncope (or asphyxia?).

Another branch of physiological evidence unexpectedly presented itself. The deceased, during her fatal illness, had vomited on the floor of her room. An alcoholic extract was made of the scrapings of the floor and of the substances deposited between the planks. No mineral poison was found in it. Seventy-five grains of this extract were introduced into the cellular membrane of the thigh of a dog. The animal suffered from vomiting and depression of the action of the heart, and died in about twenty-two hours. There was no coma or insensibility at any time. Thirty-one grains of the same extract diffused in water were administered to a rabbit by means of a funnel. In less than three hours after the injection the animal died, having suffered from an irregular and depressed action of the heart. Sixty grains of an alcoholic extract from the scrapings of the floor, said to be free from vomited matters, had no effect upon an animal.

These two extracts of the floor had different chemical properties. The first, containing, as it was believed, a portion of the vomited matters, amounted to half an ounce. It was of a brown color, had a rancid oily odor, and a bitter taste. Its solution was precipitated by tannic acid ; it was colored purple-red by sulphuric, and green by hydrochloric acid. The second was colored, had an oily aspect, but no bitterness. It was not precipitated by tannic acid, and was feebly colored by the sulphuric and hydrochloric acids; the results being different from those obtained with the first extract. It was objected to any inferences from the properties of these extracts, that the deceased's room had been formerly occupied by a photographic artist; but it is expressly stated that no noxious mineral substances, such as are used in photography, were found in them. No attempt was made to procure digitalin from the extracts; the presence of this principle in the extracts produced, was a matter of inference, and the reason assigned for the extract derived from the stomach and bowels of deceased having no fatal effect upon animals, was that the quantity of the principle left in the body at the time of death was too small.

MM. Tardieu and Roussin deposed at the trial that the deceased had died from a vegetable poison which produces no marked change in the body, which cannot be revealed by chemical analysis, but only by its noxious effects on animals. The effects on animals were in this case similar to those caused by digitalin, and without positively affirming that the deceased woman, Pauw, had died from this poison, there was the strongest presumption that she fell a victim to it. The deceased was quite well the day before her death, and the post-mortem examination of the body proved the absence of any natural cause to account for her sudden death.

In reference to the accused, it was proved that he had in his possession a large number of poisons of a deadly kind, including digitalin; that he had at three different times purchased as much as fifty-two grains of this poison, of which much had been used, and that those quantities were inconsistent with any reasonable medical requirements. As the prisoner was a homœopathic practitioner, the purchase and actual use of such large quantities of so potent a drug were quite inexplicable on any theory consistent with his innocence. On the other hand, the case was equally against him in its moral aspects; it was clearly established that, by reason of the large insurances effected on her life, he had a strong motive in the death of the woman; that a long cessation of their intimacy had taken place by reason of his marriage with another person; that he had suddenly, and without any reasonable grounds, renewed his intimacy with the deceased; and the date of her fatal illness was in accordance with these visits thus renewed. In short, Dr. De la Pommerais had the motive, means, and opportunity of destroying the life of this woman by poison, and no theory consistent with his innocence could be suggested by those who defended him, to explain satisfactorily the mass of moral and medical circumstances which were clearly proved against him. Further, as with some other criminals, he overacted his part, and by forged letters and correspondence had shown that he had fully anticipated the sudden death of the woman Pauw, and the explanations that might be required of him in order to account for this event. Apart from any questions respecting the speculative character of the medical evidence, there were circumstances proved in this case which were inconsistent with any theory of the innocence of the accused. The jury found him guilty of murder, and he was subsequently executed.

TOBACCO (NICOTIANA TABACUM).

Symptoms.—The leaves of this plant variously prepared, either as tobacco or snuff, exert a powerful action on the body, but fatal cases of poisoning by tobacco are by no means common. The effects which this substance produces, when taken in a large dose, either in the form of powder or infusion, are well-marked. The symptoms are faintness, nausea, vomiting, giddiness, delirium, loss of power in the limbs, general relaxation of the muscular system, trembling, complete prostration of strength, coldness of the surface, with cold clammy perspiration, convulsive movements, paralysis, and death sometimes by syncope. In some cases there is violent pain in the abdomen, with purging; in others there is rather a sense of sinking or depression in the region of the heart, passing into syncope, and creating a sense of impending dissolution. With the abovementioned symptoms there is dilatation of the pupils, with insensibility to light, dimness of sight, with confusion of ideas, a small, weak, and scarcely perceptible pulse, difficulty of breathing, and involuntary discharge of urine.

These symptoms indicate that tobacco may act as an irritant poison, although it more directly affects the brain, spinal marrow, and heart. There is some difficulty in assigning its true place in the classification of poisons, but it appears chiefly to affect the brain and heart, and I have, therefore, placed it among cerebral and cardiac poisons.

Its specific action on the heart has been long known to surgeons. Owing to the faintness which it rapidly produces, and the complete muscular relaxation which follows, tobacco was formerly used in infusion and decoction in cases of strangulated hernia. This enabled a surgeon to effect the reduction of the hernia, but in some unrecorded cases at the expense of the life of his patient.

In Oct. 1855, a lunatic sailor swallowed from half an ounce to one ounce of crude tobacco, having, it is believed, kept it for some time in his mouth before he swallowed it. This was not known at the time of his admission into the asylum. After he had been placed in a warm bath he suddenly became insensible and motionless, the whole of the muscles were relaxed, the only indications of life being a feeble respiration, and a pulse scarcely perceptible. The pupils of the eyes were strongly contracted. In half an hour violent convulsions of a tetanic kind affected the limbs. There was profuse purging, and in the fluid evacuations, some shreds of tobacco were found. This led to the knowledge that the patient had probably swallowed the poison. The stomach-pump was used with slight amendment; the pupils became dilated; the symptoms, however, returned; there was vomiting as well as purging of mucus and blood, with loud cries. The convulsions recurred with brief remissions, the limbs being at intervals rigidly flexed upon the body; and there was grinding of the teeth. The pulse was feeble and rapid—scarcely perceptible; the action of the heart was very irregular. The pupils were again contracted and insensible to light. These symptoms continued until the patient died in a fatal syncope about seven hours after his admission. (*Ed. Med. Jour.*, 1855–6, vol. 1, p. 643.) For three cases of poisoning by tobacco in which the persons recovered, see Reil, *Jour. für Toxikol.*, 1857, 2 H., p. 568. The following is from the *Naval Medical Reports:* A sailor boy had been frequently punished for chewing tobacco, and had often complained of debility, giddiness, and faintness, which were traced to the poisonous effects of this substance. On two occasions he had swallowed a piece to avoid detection. On the night of his death he went to his hammock telling his messmates that he felt sick. About ten minutes afterwards the occupant of the next hammock heard him breathing stertorously, and immediately tried to awaken him. He could not succeed, and when the surgeon came he was found to be moribund. The pupils were insensible to the influence of light; and the pulse, which was scarcely perceptible, in three minutes ceased to beat. On a post-mortem examination two small pieces of tobacco were found in the stomach. (*Brit. Med. Jour.*, 1873, p. 520.)

Tobacco acts *locally* as a poison; thus, when applied to wounded, abraded, or diseased surfaces, in the form of powder, juice, or as a decoction of the leaves, it may occasion the most alarming symptoms, and even death. (Orfila, vol. 2, p. 404; also Pereira, *Mat. Med.*, vol. 2, part 1, p. 579.) This fact is of importance, as some quack remedies for skin-diseases are composed of tobacco. A woman applied some leaves of tobacco to ulcers upon her legs. After some hours she suffered from sickness, dimness of vision, and cramps in the legs, with great prostration; she also complained of a benumbed feeling. On the third day there was great sleepiness, with headache and an irregular action of the heart. In about a week she recovered her usual health. (*Lancet*, 1871, vol. 2, p. 663.) Dr. Namias relates an instance of a smuggler being poisoned by reason of his having covered his skin with tobacco-leaves with a view of defrauding the revenue. The leaves, moistened by perspiration, produced all the effects of poisoning. The pulse was small and feeble; there was faintness attended with cold sweats. The operation of the poison seemed to be principally on the heart. M. Decaisne has observed in persons who have smoked tobacco excessively, a sedative action on the heart, indicated by intermission of the cardiac pulsations as well as those of the radial artery. (*Ed. Monthly Journal*, Aug. 1864, p. 172.)

The Vapor.—Some doubt has existed whether the *vapor* of this substance, in tobacco manufactories, is or is not injurious to the health of the workmen employed. M. Parent-Duchâtelet considered that, after a time, it had no influence on health. Subsequent researches by M. Melier have, however, led to the conclusion that the vapors long respired are injurious to health. The primary effects are headache, nausea, languor, loss of appetite, and sleep; the secondary effects are manifested by a general disturbance of the health. He attributes these symptoms to the nicotina which is volatilized. (*Gaz. Méd.*, Mai 3, 1845.)

Appearances.—There have been but few instances in which the bodies of persons, poisoned by tobacco, have been inspected. In a case reported by Mr. Eade, a girl, æt. 18, injected as a clyster a decoction made by boiling *three drachms* of common shag tobacco in a pint of water. In half an hour she complained of faintness and feeling sick, and in another half hour she became quite collapsed, with cold sweats; she vomited, was slightly convulsed, and died in an hour and a half from the time at which she injected the clyster. On inspection, the heart was found very flaccid; there were three drachms of black fluid blood in the ventricles. The stomach contained food, but had no unusual appearance. The intestines presented no trace of inflammation or redness in any part, and there was no smell of tobacco (thirty-six hours after death) either in the intestines or in any part of the body. The head was not examined. (*Med. Gaz.*, vol. 44, p. 823.) In the body of the lunatic whose case has been described (*supra*), the following appearances were met with forty hours after death. Cadaveric rigidity was very strongly developed. There was some congestion of the substance of the brain and the upper part of the spinal marrow (pons varolii and medulla oblongata). The lungs presented no unusual appearance. The heart was empty, small, and contracted. In the abdomen the liver and kidneys were much congested. On the mucous coat of the stomach there were several red patches. The intestines were contracted throughout and contained no fecal matter. The mucous membrane was of a red color, partially abraded; the intestines contained a mucous fluid tinged with blood. The mesenteric veins were distended with dark fluid blood. The bladder was contracted and empty. The blood was everywhere dark and liquid. (*Ed. Med. Jour.*, 1855–6, vol. 1, p. 643.) Orfila found on examining the body of a dog killed by this substance, that the mucous membrane of the stomach was strongly reddened throughout.

Fatal Dose and Period of Death.—Dr. McGregor has seen some of the most severe symptoms follow the administration of an injection which contained only *half a drachm* of tobacco in the form of decoction. (*Lancet*, Aug. 30, 1845, p. 240.) Dr. Paris witnessed a case, that proved rapidly fatal, in which a decoction of tobacco had been used as an injection in the attempted reduction of strangulated hernia (*Med. Jur.*, vol. 2, p. 418); and several instances of a similar kind are recorded by other writers. Dr. Pereira considers that it would not be safe to use more than fifteen or twenty grains under these circumstances, and he quotes an instance from Dr. Copland, in which death was caused by an infusion of *thirty grains*. (*Mat. Med.*, vol. 2, part 1, p. 579.) The fatal effects of tobacco may follow very speedily on its administration. Death has been known to take place in so short a period as three-quarters of an hour; and a case which occurred to M. Tavignot is reported to have terminated fatally in *eighteen minutes.* (*Brit. and For. Med. Rev.*, No. 24, p. 562.)

Chronic Poisoning.—Tobacco is rarely administered medicinally in substance. In a dose of five or six grains, *snuff* acts as a powerful emetic, and in larger doses it produces symptoms of poisoning. It is a remarkable instance of the effect of habit, that the quantity thrust into the nostrils, as a sort of morbid luxury, does not appear to produce any directly noxious effects on the system. The diurnal allowance of many snuff-takers, introduced into the rectum in powder, would most probably give rise to serious symptoms, in one whose system was not habituated to the use of tobacco. The same may be observed of the practice of chewing and of *smoking* tobacco; in the latter case, the volatile oil of tobacco, as well as nicotina, are brought into immediate contact with the mucous membrane, producing faintness, giddiness, and sickness in those not accustomed to the practice. In two instances, in which a large quantity of tobacco was consumed by smoking, death was the result. This involves a question as to the *chronic* form of poisoning by tobacco. Drs. Prout, Laycock, and Wright consider that habitual smoking is injurious to health, because it is liable to disorder the digestive functions. This is denied by others, on account of the difficulty of showing that the health of inveterate smokers is damaged by the habit, or that their lives are shortened by it. Dr. Prout's view appears to me, notwithstanding, quite reasonable. A poisonous substance like tobacco, whether in powder, juice, or vapor, cannot be brought frequently in contact with an absorbing surface like mucous membrane, without in many cases producing disorder of the system, which the consumer is probably quite ready to attribute to any other cause than that which would render it necessary for him to deprive himself of what he considers not merely a luxury, but an article actually necessary to his existence. The argument that cases cannot be adduced to show direct injury to health, proves too much, for a similar observation may be made of the habit of opium-eating. (See *Med. Gaz.*, vol. 38, p. 590, and *Lancet*, 1845, p. 240.) Controversial papers in reference to the beneficial and evil effects of smoking have been published in the medical journals, but nothing new has been elicited. There can be but little doubt that many dyspeptic as well as nervous disorders proceed from the inordinate use of tobacco in smoking (*Chemist*, Jan. 1856, p. 246); but the lovers of the narcotic, whether medical or non-medical, will never admit it.

This subject was brought before the British Medical Association, at Norwich, in August, 1874, by Dr. Drysdale, in reference to its influence on public health. (*Brit. Med. Jour.*, Sept. 1874, p. 318.) In the course of his practice he had met with many cases of disease, which he believed to be entirely due to the use of tobacco. He charged tobacco with causing blindness, palpitation of the heart, paralysis, diarrhœa, and diseases of the teeth and mucous membrane of the mouth and tongue. In the course of one week he met with two cases of complete blindness in men, entirely due, he was sure, to the use of tobacco. One of these patients was of the age of 27, and had been a most extensive smoker for some six years, consuming, he said, an ounce of Virginia tobacco daily. The other was only 24 years old, and he had been in the habit of chewing constantly, as well as smoking. His amaurosis was quite similar in its character with that of the other patient. Affections of the gums and tongue were very frequently seen in old smokers. The tongue looked as if it had been painted with a solution of nitrate of silver in some cases; in others there was lividity of the gums and great duskiness of the fauces. Dyspepsia and diarrhœa were more frequently caused by smoking than many believed, and the use of tobacco disposed to palpitation of the heart, prolapse of the rectum, etc. Whatever might be thought of this view, he could cordially subscribe to the opinion which ascribed to tobacco many of the cases of *malaise* and cachexia of men who would otherwise be in excellent health. I agree with Dr. Drysdale in thinking that the habit is productive of many nervous diseases, which those who indulge in tobacco uniformly persist in assigning to other causes.

Poisoning with tobacco has not often given rise to medico-legal discussion. This is the more remarkable, as it is an easily accessible poison, and the possession of it would not, as in the case of other substances, excite surprise or suspicion. In June, 1854, a man was charged with the death of an infant, æt. 10 weeks, by poisoning it with tobacco. He placed a quantity of tobacco in the mouth of the infant, with the view, as he stated, of making it sleep. The infant was completely narcotized, and died on the second day. It is probably more extensively used to aid the purposes of robbers than is commonly believed; and there is reason to suppose that porter and other liquors sold in brothels are sometimes drugged, either with tobacco or with snuff prepared from it. Scotch snuff is said to be used for this purpose. Dr. Ogston communicated a case of this kind to Sir R. Christison (*op. cit.*, p. 850), in which tobacco was administered to a man in whisky, and he soon afterwards died in a state of insensibility, without being able to give any account of the circumstances. Dr. Ogston detected *nicotina* in the contents of the stomach. An investigation took place in this metropolis, in the autumn of 1847, in which a man was charged with attempting to poison his wife by administering snuff in ale. The woman's life was saved by the speedy use of the stomach-pump. The case was dismissed, as there was a want of clear proof of criminal intention. A question here arose as to what quantity of tobacco would destroy life. The medical witness is reported to have said, that a quarter of an ounce infused in a pint of liquid, would be sufficient to destroy three persons. This is no doubt true. Thirty grains have proved fatal, and twenty grains might even kill an adult, but some allowance must here be made for the effects of habit. Many kinds of snuffs are, however, extensively adulterated with various powders; some contain lime, and even red lead; hence they are not to be regarded as consisting of pure tobacco.

Treatment.—The removal of the poison from the stomach by the stomach-pump or by emetics, if the poison itself does not cause vomiting; the patient should be kept in the recumbent position. Injections may be used to clear out the large intestines.

Analysis.—Tobacco may be found in substance in an organic liquid, or in the stomach; it may then be recognized by its odor and physical and botanical properties. Its poisonous effects are due to the presence of a peculiar volatile alkaloid, which, like conia, is liquid. It is called *Nicotina.* The proportion contained in dry tobacco has been determined by Schlössing and others. In 100 parts of the tobacco of Virginia he found 6.87 of Nicotina; Kentucky, 6.09; French, 4.94 to 7; Maryland, 2.29; Havana, less than 2. (*Schwarzkopf Org. Alk.*, 164.) In dry snuff the proportion is 2 per cent., and in moist snuff 1.3 per cent. Melsens has found this poisonous alkaloid in tobacco-smoke with the common products of combustion. (*Pflanzenstoffe*, Husemann, 1871, p. 457.)

NICOTINA.

This is the poisonous alkaloid of tobacco. It is a deadly poison, like prussic acid, destroying life in small doses and with great rapidity. It also resembles prussic acid in the fact that it is a compound of carbon, nitrogen, and hydrogen, and it contains no oxygen. I found that a rabbit was killed by a single drop in three minutes and a half. In fifteen seconds the animal lost all power of standing, was violently convulsed in its fore and hind legs, and its back was arched convulsively (opisthotonos). A frothy alkaline mucus escaped from its mouth having the odor of nicotina. (*Guy's Hosp. Reports*, Oct. 1858, p. 355.) A trial for murder by poisoning with this alkaloid occurred in Belgium, in 1851. (*Ann. d'Hyg.*, 1851, vol. 2, pp. 167 and 147.) The Count and Countess Bocarmé were charged with the murder of the countess's brother, a M. Fougnies, by administering to him nicotina while he was dining with them at the Château of Bitremont. The poison was forcibly administered. The deceased did not survive more than five minutes, and was not seen living by any one of the attendants. The possession of the poison, as well as the moral evidence, fixed the crime on the Count, and he was condemned and executed. The appearances after death were to a great extent altered or destroyed by the pouring of some strong acid (acetic) into the mouth and over the body of the deceased, in order to conceal or remove the odor of nicotina. M. Stas detected the poison in small quantity in the tongue, throat, stomach, liver, and lungs of the deceased, as well as in a wooden plank of the floor near to which he was sitting. A second case of poisoning and the only case recorded in this country, occurred in London as an act of suicide in June, 1858. A gentleman swallowed a quantity of nicotina from a bottle, and almost immediately afterwards was seen in the act of falling to the floor. He was carried to an adjoining room, but before this could be reached, he was dead. The symptoms noticed were that deceased stared wildly; there were no convulsions, and he died quietly, heaving a deep sigh in expiring. The symptoms therefore resembled those of prussic acid. The quantity of nicotina taken could not be determined. The deceased appears to have been rendered immediately insensible, and to have died in from three to five minutes after taking the poison. The *appearances* observed were a general relaxation of the muscles, prominent and staring eyes, bloated features, great fulness, with lividity about the neck. There was no odor resembling nicotina or tobacco perceptible about the body. When examined between two and three days after death, putrefaction had occurred, especially in the course of the veins. The swelling of the neck was found to arise from an effusion of dark liquid blood. The scalp, as well as the membranes of the brain were filled with dark-colored blood. The lungs were engorged and of a dark purple color. The cavities of the heart were empty, with the exception of the left auricle, which contained two drachms of dark-colored blood. The stomach contained a chocolate-colored fluid (reserved for analysis); the mucous membrane was of a dark crimson red color as a result of the most intense congestion. There was no odor excepting that of putrefaction. The liver was also congested and of a purple-black color. The blood throughout the body was black and liquid; but in some parts it had the consistency of treacle. I found nicotina in small quantity in the contents of the stomach, also in the liver and lungs; but as these organs had been placed in contact with the stomach, it could not be inferred that the poison had been absorbed and deposited in them.

Analysis.—A sample of nicotina which I examined had a pale amber color, and evolved a peculiar acrid odor, affecting the nose and eyes, resembling stale tobacco-smoke. It had the consistency of a thin oil, gave a greasy stain to paper, which soon disappeared, owing to its volatility. When heated on platinum or on paper it burnt with a bright-yellow flame, emitting a thick black smoke. It was powerfully alkaline, and imparted a strong alkaline reaction to water without readily dissolving in it. The aqueous solution, even when much diluted, retained the peculiar odor. Nicotina is dissolved by alcohol and ether, and the latter liquid will remove it from its aqueous solution. 1. Chloride of platinum produced in the aqueous solution an orange-yellow crystalline precipitate. 2. Corrosive sublimate, a white precipitate. 3. Arsenio-nitrate of silver, a yellow precipitate. In these three characters nicotina resembles ammonia; the differences, apart from the odor, which is an important distinction, are: 4. Iodine-water gives a brown precipitate (in ammonia there is no precipitate, the color is discharged). 5. Tannic acid gives a whitish yellow precipitate (in ammonia there is no precipitate, but a red color is imparted). 6. Chloriodide of potassium and mercury copiously precipitates it even when the solution is much diluted. 7. Gallic acid gives no precipitate (in ammonia it produces a pinkish red color, rapidly changing to an olive green). 8. Sulphuric acid and bichromate potash produce a green color by the liberation of oxide of chromium. (See *Guy's Hosp. Reports*, Oct. 1858, p. 354.)

Organic Mixtures.—To separate nicotina from the contents of the stomach, these should be digested in cold distilled water, acidulated with sulphuric acid in the proportion of a drop to an ounce. This liquid is strained, filtered, and the residue pressed. It is then to be evaporated to one-half in a water-bath—digested with its bulk of cold alcohol, filtered, and the alcoholic liquid evaporated in a water-bath. The sulphate of nicotina is now dissolved out of the residue by a small quantity of water; the solution is rendered alkaline by potash and then shaken in a tube with its bulk of ether; the ethereal liquid is allowed to evaporate in a series of watch-glasses, and if nicotina is present, the alkaloid will be left in small oily-looking globules. The odor may not be perceptible until the residue is heated, when its peculiar acridity will be brought out. A few drops of water should be added to the residue in each glass; it will then be found to be strongly alkaline; and the different tests may be applied. It was by this process that I discovered the poison in the body of the gentleman whose case is above related. In reference to the rabbit killed by a single drop (*supra*), nicotina was found in the stomach and its contents; there was a trace found in half an ounce of the blood of the animal, and the poison was clearly detected after a week in the tongue and soft parts of the throat of the animal, but there was no trace of nicotina in the liver, heart, or lungs.

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CHAPTER I. Inflammation. II. Treatment of Inflammation. III. Operations in general: Anæsthetics. IV. Minor Surgery. V. Amputations. VI. Special Amputations. VII. Effects of Injuries in General: Wounds. VIII. Gunshot Wounds. IX. Injuries of Bloodvessels. X. Injuries of Nerves, Muscles and Tendons, Lymphatics, Bursæ, Bones, and Joints. XI. Fractures. XII. Special Fractures. XIII. Dislocations. XIV. Effects of Heat and Cold. XV. Injuries of the Head. XVI. Injuries of the Back. XVII. Injuries of the Face and Neck. XVIII. Injuries of the Chest. XIX. Injuries of the Abdomen and Pelvis. XX. Diseases resulting from Inflammation. XXI. Erysipelas. XXII. Pyæmia. XXIII. Diathetic Diseases: Struma (including Tubercle and Scrofula); Rickets. XXIV. Venereal Diseases; Gonorrhœa and Chancroid. XXV. Venereal Diseases continued: Syphilis. XXVI. Tumors. XXVII. Surgical Diseases of Skin, Areolar Tissue, Lymphatics, Muscles, Tendons, and Bursæ. XXVIII. Surgical Disease of Nervous System (including Tetanus). XXIX. Surgical Diseases of Vascular System (including Aneurism). XXX. Diseases of Bone. XXXI. Diseases of Joints. XXXII. Excisions. XXXIII. Orthopædic Surgery. XXXIV. Diseases of Head and Spine. XXXV. Diseases of the Eye. XXXVI. Diseases of the Ear. XXXVII. Diseases of the Face and Neck. XXXVIII. Diseases of the Mouth, Jaws, and Throat. XXXIX. Diseases of the Breast. XL. Hernia. XLI. Special Herniæ. XLII. Diseases of Intestinal Canal. XLIII. Diseases of Abdominal Organs, and various operations on the Abdomen. XLIV. Urinary Calculus. XLV. Diseases of Bladder and Prostate. XLVI. Diseases of Urethra. XLVII. Diseases of Generative Organs. INDEX.

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